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* * * * * * * * * *   Welcome to STN International   * * * * * * * * * *

 NEWS  1           Web Page for STN Seminar Schedule - N. America
 NEWS  2  JUL 02   LMEDLINE coverage updated
 NEWS  3  JUL 02   SCISEARCH enhanced with complete author names
 NEWS  4  JUL 02   CHEMCATS accession numbers revised
 NEWS  5  JUL 02   CA/CAplus enhanced with utility model patents from China
 NEWS  6  JUL 16   CAplus enhanced with French and German abstracts
 NEWS  7  JUL 18   CA/CAplus patent coverage enhanced
 NEWS  8  JUL 26   USPATFULL/USPAT2 enhanced with IPC reclassification
 NEWS  9  JUL 30   USGENE now available on STN
 NEWS 10  AUG 06   CAS REGISTRY enhanced with new experimental property tags
 NEWS 11  AUG 06   BEILSTEIN updated with new compounds
 NEWS 12  AUG 06   FSTA enhanced with new thesaurus edition
 NEWS 13  AUG 13   CA/CAplus enhanced with additional kind codes for granted
                   patents
 NEWS 14  AUG 20   CA/CAplus enhanced with CAS indexing in pre-1907 records
 NEWS 15  AUG 27   Full-text patent databases enhanced with predefined
                   patent family display formats from INPADOCDB
 NEWS 16  AUG 27   USPATOLD now available on STN
 NEWS 17  AUG 28   CAS REGISTRY enhanced with additional experimental
                   spectral property data
 NEWS 18  SEP 07   STN AnaVist, Version 2.0, now available with Derwent
                   World Patents Index
 NEWS 19  SEP 13   FORIS renamed to SOFIS
 NEWS 20  SEP 13   INPADOCDB enhanced with monthly SDI frequency
 NEWS 21  SEP 17   CA/CAplus enhanced with printed CA page images from
                   1967-1998
 NEWS 22  SEP 17   CAplus coverage extended to include traditional medicine
                   patents
 NEWS 23  SEP 24   EMBASE, EMBAL, and LEMBASE reloaded with enhancements

 NEWS EXPRESS  19 SEPTEMBER 2007:  CURRENT WINDOWS VERSION IS V8.2,
               CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 19 SEPTEMBER 2007.

 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS LOGIN    Welcome Banner and News Items
 NEWS IPC8     For general information regarding STN implementation of IPC 8

Enter NEWS followed by the item number or name to see news on that
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FILE 'HOME' ENTERED AT 10:32:29 ON 24 SEP 2007

=> File .Gerry2MBCE
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
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FULL ESTIMATED COST                                         0.21       0.21

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=> S (Stem(A)Cell)(S)(Stimulator OR differentiator) AND pd<=20040415
   2 FILES SEARCHED...
L1          220 (STEM(A) CELL)(S)(STIMULATOR OR DIFFERENTIATOR) AND PD<=20040415

=> DUP Rem L1
PROCESSING COMPLETED FOR L1
L2          103 DUP REM L1 (117 DUPLICATES REMOVED)
                ANSWERS '1-58' FROM FILE MEDLINE
                ANSWERS '59-80' FROM FILE BIOSIS
                ANSWERS '81-96' FROM FILE CAPLUS
                ANSWERS '97-103' FROM FILE EMBASE

=> S L2 AND (beta(A)cell OR Langerhan?)
L3            2 L2 AND (BETA(A) CELL OR LANGERHAN?)

=> D ibib abs L3 1,2

L3   ANSWER 1 OF 2     MEDLINE on STN
ACCESSION NUMBER:   2004626505     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 15601372
TITLE:              A perspective on pancreatic stem/progenitor cells.
AUTHOR:             Habener Joel F
CORPORATE SOURCE:   Laboratory of Molecular Endocrinology, Massachusetts
                    General Hospital, 55 Fruit Street - WEL 320, Boston, MA
                    02114, USA.. jhabener@partners.org
SOURCE:             Pediatric diabetes, (2004) Vol. 5 Suppl 2, pp.
                    29-37.  Ref: 119
                    Journal code: 100939345. ISSN: 1399-543X.
PUB. COUNTRY:       Denmark
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
                    (RESEARCH SUPPORT, NON-U.S. GOV'T)
                    (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
                    General Review; (REVIEW)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200505
ENTRY DATE:         Entered STN: 20 Dec 2004
                    Last Updated on STN: 11 May 2005
                    Entered Medline: 10 May 2005
AB   The prevalence of both type 1 and type 2 diabetes mellitus is increasing
     throughout the world along with the ensuant morbidity and early mortality
     because of premature microvascular and macrovascular disease.  Current
     insulin and drug therapies control diabetes, but do not cure it.
     Cell-based therapies offer the possibilities of a permanent cure for
     diabetes.  Recently, success in the transplantation of pancreatic islets
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in the livers of type 1 diabetics has afforded the opportunity for a potential cure. However, the severe shortage of donor islets for transplantation limits the usefulness of this therapy. One approach is to exploit the use of stem cells, either embryo-derived or adult tissue-derived, as substrates to create islet tissue suitable for transplantation. Cells isolated from embryo blastocysts and from adult pancreas, liver, and bone marrow can be expanded extensively in vitro and differentiated into islet-like clusters that produce insulin, and, in some instances, can achieve glycemic control when transplanted into streptozotocin-induced diabetic mice. It is, now, also possible to envision the direct systemic administration of stem cells that would home in on and regenerate injured islets, or to administer stem cell stimulators that would enhance endogenous pancreatic stem cells to expand and differentiate into functional, insulin-producing beta-cells. This perspective discusses the potential applications of cellular medicines, in the new discipline of regenerative medicine, to achieve a cure for diabetes.

L3 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:837240 CAPLUS
DOCUMENT NUMBER: 139:335101
TITLE: Method for isolating and measuring proliferation of long-term label retaining cells and stem cells
INVENTOR(S): Hellerstein, Marc K.; Kim, Sylvia Jeewon
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: PCT Int. Appl., 62 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2003087314	A2	20031023	WO 2003-US10554		20030404 <--
WO 2003087314	A3	20050224			
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
CA 2503681	A1	20031023	CA 2003-2503681		20030404 <--
AU 2003234688	A1	20031027	AU 2003-234688		20030404 <--
US 2003224420	A1	20031204	US 2003-407435		20030404 <--
PRIORITY APPLN. INFO.:			US 2002-370599P	P	20020405
			WO 2003-US10554	W	20030404

AB This invention relates to a method for separating long-term label retaining cells or stem cells. In particular, this invention relates to a method for separating long-term label retaining cells and/or stem cells from tissues or individuals and for measuring proliferation rates of long-term label retaining cells and stem cells, as well as determining clonal expansion (proliferative history) of cell lineages from the tissues of the individual. The cells may be double-labeled with a cell-lineage marking label and isotopically labeled DNA synthesis precursor prior to phys. separation A double-labeling approach was developed using bromodeoxyuridine (BrdU) as a marker for label retaining cells and 2H2O to determine their proliferation rates using gas chromatog.-mass spectrometry. Rats were given BrdU in drinking water and 2H2O i.p. and in drinking water. Label-retaining cells were isolated from colon epithelial cells and

collected by FACS. The DNA was isolated, hydrolyzed, and the pentose-tetraacetate derivative of the deoxyribose of dA was prepared for GC/MS anal. for determining cell proliferation.

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STN INTERNATIONAL SESSION SUSPENDED AT 10:39:05 ON 24 SEP 2007
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FILE 'BIOSIS' ENTERED AT 12:19:50 ON 24 SEP 2007
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FULL ESTIMATED COST	27.32	27.53

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-0.78	-0.78

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FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 10:32:53 ON 24 SEP 2007
L1 220 S (STEM(A)CELL)(S)(STIMULATOR OR DIFFERENTIATOR) AND PD<=200404
L2 103 DUP REM L1 (117 DUPLICATES REMOVED)
L3 2 S L2 AND (BETA(A)CELL OR LANGERHAN?)

=> D Ti L2 1-103

L2 ANSWER 1 OF 103 MEDLINE on STN DUPLICATE 1
TI Extracellular nucleotides are potent stimulators of human hematopoietic stem cells in vitro and in vivo.

L2 ANSWER 2 OF 103 MEDLINE on STN DUPLICATE 2
TI Improved detection of clinically significant host-reactive antigens prior to HLA-identical sibling peripheral blood stem cell transplantation using a dendritic cell-based helper T-lymphocyte precursor assay.

L2 ANSWER 3 OF 103 MEDLINE on STN DUPLICATE 3
TI A perspective on pancreatic stem/progenitor cells.

L2 ANSWER 4 OF 103 MEDLINE on STN DUPLICATE 4
TI Human CD34+ hematopoietic stem cells capable of multilineage engrafting

NOD/SCID mice express flt3: distinct flt3 and c-kit expression and response patterns on mouse and candidate human hematopoietic stem cells.

L2 ANSWER 5 OF 103 MEDLINE on STN DUPLICATE 5
TI The competitive nature of HOXB4-transduced HSC is limited by PBX1: the generation of ultra-competitive stem cells retaining full differentiation potential.

L2 ANSWER 6 OF 103 MEDLINE on STN DUPLICATE 7
TI Dendritic cells (DCs) in rheumatoid arthritis (RA): progenitor cells and soluble factors contained in RA synovial fluid yield a subset of myeloid DCs that preferentially activate Th1 inflammatory-type responses.

L2 ANSWER 7 OF 103 MEDLINE on STN DUPLICATE 8
TI Management of osteochondral injuries of the knee.

L2 ANSWER 8 OF 103 MEDLINE on STN DUPLICATE 9
TI High-resolution tracking of cell division suggests similar cell cycle kinetics of hematopoietic stem cells stimulated in vitro and in vivo.

L2 ANSWER 9 OF 103 MEDLINE on STN DUPLICATE 10
TI Stromal cell-derived factor-1 (SDF-1) acts together with thrombopoietin to enhance the development of megakaryocytic progenitor cells (CFU-MK).

L2 ANSWER 10 OF 103 MEDLINE on STN DUPLICATE 11
TI Identification of cord blood dendritic cells as an immature CD11c- population.

L2 ANSWER 11 OF 103 MEDLINE on STN DUPLICATE 12
TI Expression and regulation of the thrombopoietin receptor variants MPLP and MPLK in PBMC.

L2 ANSWER 12 OF 103 MEDLINE on STN DUPLICATE 13
TI Cord blood mononuclear cell transformation assay for screening for the presence of Epstein-Barr virus.

L2 ANSWER 13 OF 103 MEDLINE on STN DUPLICATE 14
TI The in vitro effects of all-trans-retinoic acid and hematopoietic growth factors on the clonal growth and self-renewal of blast stem cells in acute promyelocytic leukemia.

L2 ANSWER 14 OF 103 MEDLINE on STN DUPLICATE 15
TI Studies of a 35 KDa substance from human fetal liver on the regulation of hematopoiesis.

L2 ANSWER 15 OF 103 MEDLINE on STN DUPLICATE 16
TI The FLT3 ligand is a direct and potent stimulator of the growth of primitive and committed human CD34+ bone marrow progenitor cells in vitro.

L2 ANSWER 16 OF 103 MEDLINE on STN DUPLICATE 17
TI The FLT3 ligand potently and directly stimulates the growth and expansion of primitive murine bone marrow progenitor cells in vitro: synergistic interactions with interleukin (IL) 11, IL-12, and other hematopoietic growth factors.

L2 ANSWER 17 OF 103 MEDLINE on STN DUPLICATE 18
TI The molecular specificity of action of the tetrapeptide acetyl-N-Ser-Asp-Lys-Pro (AcSDKP) in the control of hematopoietic stem cell proliferation.

L2 ANSWER 18 OF 103 MEDLINE on STN DUPLICATE 19
TI Cytotoxic lymphocyte maturation factor (interleukin 12) is a synergistic growth factor for hematopoietic stem cells.

L2 ANSWER 19 OF 103 MEDLINE on STN DUPLICATE 20

TI The in vivo effects of steel factor on natural killer lineage cells in murine spleen and bone marrow.

L2 ANSWER 20 OF 103 MEDLINE on STN DUPLICATE 21
TI Ways of minimising hematopoietic damage induced by radiation and cytostatic drugs--the possible role of inhibitors.

L2 ANSWER 21 OF 103 MEDLINE on STN DUPLICATE 22
TI Inhibitory effects of AcSDKP on the mixed lymphocyte reaction (MLR). Part I. MLR with mouse spleen cells.

L2 ANSWER 22 OF 103 MEDLINE on STN DUPLICATE 23
TI The mechanism of action of the tetrapeptide acetyl-N-Ser-Asp-Lys-Pro (AcSDKP) in the control of haematopoietic stem cell proliferation.

L2 ANSWER 23 OF 103 MEDLINE on STN DUPLICATE 24
TI Protection from arabinofuranosylcytosine and n-mustard-induced myelotoxicity using hemoregulatory peptide pGlu-Glu-Asp-Cys-Lys monomer and dimer.

L2 ANSWER 24 OF 103 MEDLINE on STN DUPLICATE 25
TI Haemoprotection against cytostatic drugs by stem cell inhibition.

L2 ANSWER 25 OF 103 MEDLINE on STN DUPLICATE 26
TI Haematopoietic stem cell proliferation regulators investigated using an in vitro assay.

L2 ANSWER 26 OF 103 MEDLINE on STN DUPLICATE 27
TI [The regulation of hematopoietic stem cell proliferation and differentiation (CFU-S) during antigenic exposure]. Reguliatsiia proliferatsii i differentsirovki gemopoeticheskikh stvolovykh kletok (KOEs) pri antigennom vozdeistvii.

L2 ANSWER 27 OF 103 MEDLINE on STN DUPLICATE 28
TI Regulation of haematopoietic stem cell proliferation by stimulatory factors produced by murine fetal and adult liver.

L2 ANSWER 28 OF 103 MEDLINE on STN DUPLICATE 29
TI Enhanced myelopoiesis in long-term cultures of human bone marrow pretreated with recombinant granulocyte-macrophage colony-stimulating factor.

L2 ANSWER 29 OF 103 MEDLINE on STN DUPLICATE 30
TI The effects of recombinant CSF-1 on the blast cells of acute myeloblastic leukemia in suspension culture.

L2 ANSWER 30 OF 103 MEDLINE on STN DUPLICATE 31
TI A stimulator of mouse stem cell proliferation produced by human regenerating bone marrow.

L2 ANSWER 31 OF 103 MEDLINE on STN DUPLICATE 32
TI Production of human pluripotent progenitor cell colony stimulating activity (CFU-GEMMCSA) in patients with myelodysplastic syndromes.

L2 ANSWER 32 OF 103 MEDLINE on STN DUPLICATE 33
TI A stimulator of murine haemopoietic stem cell proliferation produced by human fetal liver cells.

L2 ANSWER 33 OF 103 MEDLINE on STN DUPLICATE 34
TI Controls on the cell cycle.

L2 ANSWER 34 OF 103 MEDLINE on STN DUPLICATE 35
TI Quantitative problems in bone marrow transplantation by peripheral blood stem cells.

L2 ANSWER 35 OF 103 MEDLINE on STN DUPLICATE 36
TI Vitamin C and thiol reagents promote the in vitro growth of murine granulocyte/macrophage progenitor cells by neutralizing endogenous inhibitor(s).

L2 ANSWER 36 OF 103 MEDLINE on STN DUPLICATE 37
TI Comparison of haemopoiesis in young and old mice.

L2 ANSWER 37 OF 103 MEDLINE on STN DUPLICATE 38
TI Cyclic AMP response to various haemopoietic regulators.

L2 ANSWER 38 OF 103 MEDLINE on STN DUPLICATE 39
TI Spatial organisation of CFU-S proliferation regulators in the mouse femur.

L2 ANSWER 39 OF 103 MEDLINE on STN DUPLICATE 40
TI The cellular specificity of haemopoietic stem cell proliferation regulators.

L2 ANSWER 40 OF 103 MEDLINE on STN DUPLICATE 41
TI Effect of a neutrophilia-inducing tumor on hemopoietic stem cells in mice.

L2 ANSWER 41 OF 103 MEDLINE on STN DUPLICATE 42
TI Lithium stimulates the recovery of granulopoiesis following acute radiation injury.

L2 ANSWER 42 OF 103 MEDLINE on STN DUPLICATE 43
TI Injury and regeneration in rat small intestine cells after exposure to neutrons.

L2 ANSWER 43 OF 103 MEDLINE on STN DUPLICATE 44
TI Stimulation of haemopoietic stem cell proliferation: characteristics of the stimulator-producing cells.

L2 ANSWER 44 OF 103 MEDLINE on STN DUPLICATE 48
TI The relationship of G0 to the cell cycle of haemopoietic spleen colony-forming cells.

L2 ANSWER 45 OF 103 MEDLINE on STN DUPLICATE 49
TI The regulation of hemopoiesis in long-term bone marrow cultures. II. Stimulation and inhibition of stem cell proliferation.

L2 ANSWER 46 OF 103 MEDLINE on STN DUPLICATE 50
TI Sources of haemopoietic stem cell proliferation: stimulators and inhibitors.

L2 ANSWER 47 OF 103 MEDLINE on STN DUPLICATE 52
TI Current concepts of abnormal stem cell proliferation in human disease.

L2 ANSWER 48 OF 103 MEDLINE on STN DUPLICATE 53
TI Response of neutropenia and anaemia to immunosuppressive therapy: report and bone marrow culture studies.

L2 ANSWER 49 OF 103 MEDLINE on STN DUPLICATE 55
TI Production of stem cell proliferation stimulators and inhibitors by haemopoietic cell suspensions.

L2 ANSWER 50 OF 103 MEDLINE on STN DUPLICATE 56
TI The effect of E type prostaglandins on the proliferation of haemopoietic stem cells in vivo.

L2 ANSWER 51 OF 103 MEDLINE on STN DUPLICATE 57
TI A stimulator of stem cell proliferation in regenerating bone marrow.

L2 ANSWER 52 OF 103 MEDLINE on STN DUPLICATE 58

TI Granulopoiesis in severe congenital neutropenia.

L2 ANSWER 53 OF 103 MEDLINE on STN
TI Influence of serum from liver-damaged rats on differentiation tendency of bone marrow-derived stem cells.

L2 ANSWER 54 OF 103 MEDLINE on STN
TI Autologous peripheral blood stem cell transplantation for myocardial regeneration: a novel strategy for cell collection and surgical injection.

L2 ANSWER 55 OF 103 MEDLINE on STN
TI Clinical study of single-dose G-CSF in mobilization and reconstruction of allogeneic peripheral blood stem cell transplantation.

L2 ANSWER 56 OF 103 MEDLINE on STN
TI [Effect of sera from children with acute lymphoblastic leukemia on the morphology of granulocyte-macrophage colonies grown from murine bone marrow cells and the cells of murine myelomonocytic leukemia WEHI 3B D+]. Wplyw surowic dzieci w przebiegu ostrej bialaczki limfoblastycznej na morfologie kolonii granulocytarno-makrofagowych (GM) powstalych z komorek macierzystych szpiku myszy (CFC) oraz komorek mysiej bialaczki mielomonocytarnej WEHI 3B D+.

L2 ANSWER 57 OF 103 MEDLINE on STN
TI Feedback regulators in normal and tumour tissues.

L2 ANSWER 58 OF 103 MEDLINE on STN
TI Prostaglandin E2 as stimulator of haemopoietic stem cell proliferation.

L2 ANSWER 59 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 6
TI Osteoblastic differentiation of mesenchymal stem cells by mumie extract.

L2 ANSWER 60 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 45
TI INTERFERON ITS ROLE IN RADIOPROTECTION AS A HEMATOPOIETIC STEM CELL STIMULATOR.

L2 ANSWER 61 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 46
TI INTERRELATIONSHIPS OF INHIBITOR AND STIMULATOR IN THE REGULATION OF HEMOPOIETIC STEM CELL PROLIFERATION.

L2 ANSWER 62 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 47
TI EFFECTS OF CELL CONCENTRATIONS ON THE SURVIVAL AND RE POPULATION OF HEMOPOIETIC STEM CELLS IN IRRADIATED BONE MARROW CELL CULTURE IN-VITRO.

L2 ANSWER 63 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 51
TI ACTIONS OF THE HEMOPOIETIC STEM CELL PROLIFERATION INHIBITOR.

L2 ANSWER 64 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 59
TI PROSTAGLANDIN E-2 AS STIMULATOR OF HEMOPOIETIC STEM CELL PROLIFERATION.

L2 ANSWER 65 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Influence of Glial Cell Line-Derived Neurotrophic Factor (GDNF) on Spermatogonial Stem Cells.

L2 ANSWER 66 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

TI Surgical injection of autologous, G-CSF mobilized, peripheral blood CD133+ cells for myocardial regeneration in patients undergoing coronary artery bypass grafting.

L2 ANSWER 67 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Hman CD34+ Hematopoietic Stem Cells Capable of Multilineage Engrafting NOD/SCID Mice Express Flt3: Evidence for Distinct Flt3 and C-Kit Expression and Response Patterns on Mouse and Candidate Human Stem Cells.

L2 ANSWER 68 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Non-Availability of Clinical Grade Reagents Prohibits the Clinical Application of In Vitro Cultured Peptide-Specific Cytotoxic T Lymphocytes(CTL).

L2 ANSWER 69 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Allogeneic mesenchymal stem cells persist and function in an immunocompetent non-human primate model.

L2 ANSWER 70 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Bone marrow cultures for developing suppressor and stimulator cells for research and therapeutic applications.

L2 ANSWER 71 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI An orally available small molecule stimulator of bone marrow stem cells accelerates postchemotherapy recovery of peripheral neutrophils and platelets.

L2 ANSWER 72 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI TUMOR NECROSIS FACTOR-ALPHA IS A POTENT STIMULATOR OF A VERY PRIMITIVE HEMATOPOIETIC STEM CELL IN LONG-TERM BONE MARROW CULTURES.

L2 ANSWER 73 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI ABSENCE OF HEMOPOIETIC STEM CELL PROLIFERATION INHIBITOR PRODUCTION BY BONE MARROW MACROPHAGES IN AGED MICE.

L2 ANSWER 74 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI STUDY OF CELLS PARTICIPATING IN THE PRODUCTION OF COLONY-FORMING UNITS IN THE SPLEEN USING COMBINED METHODS OF BONE MARROW FRACTIONATION.

L2 ANSWER 75 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI A STIMULATOR OF MURINE HEMOPOIETIC STEM CELL PROLIFERATION PRODUCED BY HUMAN FETAL LIVER CELLS.

L2 ANSWER 76 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI MURINE MALARIA DECREASES HEMOPOIETIC STEM CELLS AND TOTAL BONE MARROW CELLULARITY.

L2 ANSWER 77 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI EFFECT OF HEMOPOIETIC STEM-CELL PROLIFERATION REGULATORS ON EARLY AND LATE SPLEEN COLONY-FORMING CELLS.

L2 ANSWER 78 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

TI TREATMENT OF APLASTIC ANEMIA WITH METHENOLONE STANOZOLOL AND NANDROLONE 130 CASES.

L2 ANSWER 79 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI EXPERIMENTAL AND CLINICAL INVESTIGATIONS ON STEM CELL TAKE AND COLONY FORMATION.

L2 ANSWER 80 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI EFFECTS OF IRRADIATION ON STEM CELL RESPONSE TO DIFFERENTIATION INHIBITORS IN THE PLANARIAN DUGESIA-ETRUSCA.

L2 ANSWER 81 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 54
TI Production of stem cell proliferation regulators by fractionated hemopoietic cell suspensions

L2 ANSWER 82 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
TI Inhibitor and stimulator of stem cell proliferation and uses thereof

L2 ANSWER 83 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
TI Asymmetric division and lineage commitment at the level of hematopoietic stem cells: Inference from differentiation in daughter cell and granddaughter cell pairs

L2 ANSWER 84 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
TI Method for isolating and measuring proliferation of long-term label retaining cells and stem cells

L2 ANSWER 85 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
TI Tissue-restricted T cell alloresponses across HLA barriers: selection and identification of leukemia-restricted CTL in HLA-mismatched stimulator-responder pairs

L2 ANSWER 86 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
TI Quiescent CD34+ early erythroid progenitors are resistant to several erythropoietic 'inhibitory' cytokines; role of FLIP

L2 ANSWER 87 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
TI Protein and cDNA sequences of a novel chicken leukemia inhibitory factor (LIF)

L2 ANSWER 88 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
TI Small GTP-binding protein GDP dissociation stimulator gene knockout mouse for study of antiapoptotic cell survival signaling

L2 ANSWER 89 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
TI Method for regulating the differentiation/proliferation of hematopoietic stem cells with differentiation-repressing gene and blood cell-stimulators

L2 ANSWER 90 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
TI In vitro expansion of hematopoietic stem cells using an engineered hybrid cytokine of interleukin-6 and its receptor.

L2 ANSWER 91 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
TI Inhibitor and stimulator of stem cell proliferation and uses thereof

L2 ANSWER 92 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
TI Methods for introducing genes into hematopoietic stem cells in the presence of factors capable of stimulating gp130 and/or c-kit

L2 ANSWER 93 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN

TI Functions of IL-3

L2 ANSWER 94 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
TI Compositions for stimulating growth of hematopoietic stem cells committed to differentiate to megakaryocytes

L2 ANSWER 95 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
TI Purification and identification of a hematopoietic stem cell proliferation stimulator from human fetal liver

L2 ANSWER 96 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
TI Characterization of stimulatory activity for human pluripotent stem cells (CFUGEMM)

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TI Manipulation of the stem cell as a target for hematologic malignancies.

L2 ANSWER 98 OF 103 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
TI [Disturbed regulation of the stem cell proliferation in a patient with erythroblasto- and reticulocytopenia].
DYSREGULATION DER STAMMZELL-PROLIFERATION. NACHWEIS BEI EINEM PATIENTEN MIT ERYTHROBLASTO- UND RETIKULOZYTOPENIE.

L2 ANSWER 99 OF 103 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
TI Purification and biochemical characterisation of a CFU-S proliferation inhibitor: Preliminary results.

L2 ANSWER 100 OF 103 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
TI Regulation of pluripotent stem cell proliferation and differentiation: The role of long-range humoral factors.

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TI Effects of radiations on bone marrow.

L2 ANSWER 102 OF 103 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
TI [Kinetics and regulatory mechanisms of granulocyte turnover].
KINETIK UND REGULATIONSMECHANISMEN DES GRANULOZYTENUMSATZES.

L2 ANSWER 103 OF 103 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
TI Hematopoietic stem cell regulation. II. Chronic effects of hypoxic hypoxia on CFU kinetics.

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SESSION WILL BE HELD FOR 120 MINUTES
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SESSION RESUMED IN FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE'
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FILE 'MEDLINE' ENTERED AT 12:30:24 ON 24 SEP 2007
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FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 10:32:53 ON 24 SEP 2007
L1 220 S (STEM(A)CELL)(S)(STIMULATOR OR DIFFERENTIATOR) AND PD<=200404
L2 103 DUP REM L1 (117 DUPLICATES REMOVED)
L3 2 S L2 AND (BETA(A)CELL OR LANGERHAN?)

=> D ibib abs L2 3,8,9,16,19,25,27,29,30,34,43,46,49,51,58,60,66,71,79,82,95,100

L2 ANSWER 3 OF 103 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2004626505 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15601372
TITLE: A perspective on pancreatic stem/progenitor cells.
AUTHOR: Habener Joel F
CORPORATE SOURCE: Laboratory of Molecular Endocrinology, Massachusetts General Hospital, 55 Fruit Street - WEL 320, Boston, MA 02114, USA.. jhabener@partners.org
SOURCE: Pediatric diabetes, (2004) Vol. 5 Suppl 2, pp. 29-37. Ref: 119
Journal code: 100939345. ISSN: 1399-543X.
PUB. COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200505
ENTRY DATE: Entered STN: 20 Dec 2004
Last Updated on STN: 11 May 2005
Entered Medline: 10 May 2005

AB The prevalence of both type 1 and type 2 diabetes mellitus is increasing throughout the world along with the ensuant morbidity and early mortality because of premature microvascular and macrovascular disease. Current insulin and drug therapies control diabetes, but do not cure it. Cell-based therapies offer the possibilities of a permanent cure for diabetes. Recently, success in the transplantation of pancreatic islets in the livers of type 1 diabetics has afforded the opportunity for a potential cure. However, the severe shortage of donor islets for transplantation limits the usefulness of this therapy. One approach is to exploit the use of stem cells, either embryo-derived or adult tissue-derived, as substrates to create islet tissue suitable for transplantation. Cells isolated from embryo blastocysts and from adult pancreas, liver, and bone marrow can be expanded extensively in vitro and differentiated into islet-like clusters that produce insulin, and, in some

instances, can achieve glycemic control when transplanted into streptozotocin-induced diabetic mice. It is, now, also possible to envision the direct systemic administration of stem cells that would home in on and regenerate injured islets, or to administer stem cell stimulators that would enhance endogenous pancreatic stem cells to expand and differentiate into functional, insulin-producing beta-cells. This perspective discusses the potential applications of cellular medicines, in the new discipline of regenerative medicine, to achieve a cure for diabetes.

L2 ANSWER 8 OF 103 MEDLINE on STN DUPLICATE 9
ACCESSION NUMBER: 2000115167 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10648396
TITLE: High-resolution tracking of cell division suggests similar cell cycle kinetics of hematopoietic stem cells stimulated in vitro and in vivo.
AUTHOR: Oostendorp R A; Audet J; Eaves C J
CORPORATE SOURCE: Terry Fox Laboratory, British Columbia Cancer Agency, Vancouver, BC, Canada.
CONTRACT NUMBER: P01-HL55435 (NHLBI)
SOURCE: Blood, (2000 Feb 1) Vol. 95, No. 3, pp. 855-62. Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: (COMPARATIVE STUDY) Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T) (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 9 Mar 2000 Last Updated on STN: 9 Mar 2000 Entered Medline: 24 Feb 2000

AB The kinetics of proliferation of primitive murine bone marrow (BM) cells stimulated either in vitro with growth factors (fetal liver tyrosine kinase ligand 3 [FL], Steel factor [SF], and interleukin-11 [IL-11], or hyper-IL-6) or in vivo by factors active in myeloablated recipients were examined. Cells were first labeled with 5- and 6-carboxyfluorescein diacetate succinimidyl ester (CFSE) and then incubated overnight prior to isolating CFSE(+) cells. After 2 more days in culture, more than 90% of the in vivo lymphomyeloid repopulating activity was associated with the most fluorescent CFSE(+) cells (ie, cells that had not yet divided), although this accounted for only 25% of the repopulating stem cells measured in the CFSE(+) "start" population. After a total of 4 days in culture (1 day later), 15-fold more stem cells were detected (ie, 4-fold more than the day 1 input number), and these had become (and thereafter remained) exclusively associated with cells that had divided at least once in vitro. Flow cytometric analysis of CFSE(+) cells recovered from the BM of transplanted mice indicated that these cells proliferated slightly faster (up to 5 divisions completed within 2 days and up to 8 divisions completed within 3 days in vivo versus 5 and 7 divisions, respectively, in vitro). FL, SF, and ligands which activate gp130 are thus efficient stimulators of transplantable stem cell self-renewal divisions in vitro. The accompanying failure of these cells to accumulate rapidly indicates important changes in their engraftment potential independent of accompanying changes in their differentiation status.

L2 ANSWER 9 OF 103 MEDLINE on STN DUPLICATE 10
ACCESSION NUMBER: 2000115155 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10648384
TITLE: Stromal cell-derived factor-1 (SDF-1) acts together with thrombopoietin to enhance the development of megakaryocytic progenitor cells (CFU-MK).

AUTHOR: Hodohara K; Fujii N; Yamamoto N; Kaushansky K
CORPORATE SOURCE: Division of Hematology, University of Washington School of Medicine, Seattle 98195-7710, USA.
CONTRACT NUMBER: CA31615 (NCI)
DK 49855 (NIDDK)
SOURCE: Blood, (2000 Feb 1) Vol. 95, No. 3, pp. 769-75.
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 9 Mar 2000
Last Updated on STN: 9 Mar 2000
Entered Medline: 24 Feb 2000

AB Stromal cell-derived factor-1 (SDF-1) is a CXC chemokine that acts as a stimulator of pre-B lymphocyte cell growth and as a chemoattractant for T cells, monocytes, and hematopoietic stem cells. More recent studies also suggest that megakaryocytes migrate in response to SDF-1. Because genetic elimination of SDF-1 or its receptor lead to marrow aplasia, we investigated the effect of SDF-1 on megakaryocyte progenitors (colony-forming units-megakaryocyte [CFU-MK]). We report that SDF-1 augments the growth of CFU-MK from whole murine bone marrow cells when combined with thrombopoietin (TPO). The addition of SDF-1 to interleukin-3 (IL-3) or stem cell factor (SCF) had no effect. Specific antagonists for CXCR4 (the sole receptor for SDF-1), T22, and 1-9 (P2G) SDF-1 reduced megakaryocyte colony growth induced by TPO alone, suggesting that many culture systems contain endogenous levels of the chemokine that contributes to the TPO effect. To examine whether SDF-1 has direct effects on CFU-MK, we developed a new protocol to purify megakaryocyte progenitors. CFU-MK were highly enriched in CD41(high) c-kit(high) cells generated from lineage-depleted TPO-primed marrow cells. Because the growth-promoting effects of SDF-1 were also observed when highly purified populations of CFU-MK were tested in serum-free cultures, these results suggest that SDF-1 directly promotes the proliferation of megakaryocytic progenitors in the presence of TPO, and in this way contributes to the favorable effects of the bone marrow microenvironment on megakaryocyte development.

L2 ANSWER 16 OF 103 MEDLINE on STN DUPLICATE 17
ACCESSION NUMBER: 95213660 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7535335
TITLE: The FLT3 ligand potently and directly stimulates the growth and expansion of primitive murine bone marrow progenitor cells in vitro: synergistic interactions with interleukin (IL) 11, IL-12, and other hematopoietic growth factors.
AUTHOR: Jacobsen S E; Okkenhaug C; Myklebust J; Veiby O P; Lyman S D
CORPORATE SOURCE: Department of Immunology, Norwegian Radium Hospital, Oslo.
SOURCE: The Journal of experimental medicine, (1995 Apr 1) Vol. 181, No. 4, pp. 1357-63.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199505
ENTRY DATE: Entered STN: 10 May 1995
Last Updated on STN: 3 Mar 2000
Entered Medline: 2 May 1995

AB The recently cloned murine flt3 ligand (FL) was studied for its ability to stimulate the growth of primitive (Lin-Sca-1+) and more committed (Lin-Sca-1-) murine bone marrow progenitor cells, alone and in combination

with other hematopoietic growth factors (HGFs). Whereas FL was a weak proliferative stimulator alone, it potently synergized with a number of other HGFs, including all four colony-stimulating factor (CSF), interleukin (IL) 6, IL-11, IL-12, and stem cell factor (SCF), to promote the colony formation of Lin-Sca-1+, but not Lin-Sca-1- or erythroid progenitor cells. The synergistic activity of FL was concentration dependent, with maximum stimulation occurring at 250 ng/ml, and was observed when cells were plated at a concentration of one cell per culture, suggesting that its effects are directly mediated. 2 wk of treatment with FL in combination with IL-3 or SCF resulted in the production of a high proportion of mature myeloid cells (granulocytes and macrophages), whereas the combination of FL with G-CSF, IL-11, or IL-12 resulted predominantly in the formation of cells with an immature blast cell appearance. Accordingly, FL in combination with G-CSF or IL-11 expanded the number of progenitors more than 40-fold after 2 wk incubation. Thus, FL emerges as a potent synergistic HGF, that in combination with numerous other HGFs, can directly stimulate the proliferation, myeloid differentiation, and expansion of primitive hematopoietic progenitor cells.

L2 ANSWER 19 OF 103 MEDLINE on STN DUPLICATE 20
ACCESSION NUMBER: 94093292 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7505667
TITLE: The in vivo effects of steel factor on natural killer lineage cells in murine spleen and bone marrow.
AUTHOR: Miller S C; Fleming W H; Zsebo K M; Weissman I L
CORPORATE SOURCE: Department of Anatomy, McGill University, Montreal, Canada.
SOURCE: Natural immunity, (1993 Nov-Dec) Vol. 12, No. 6, pp. 293-301.
Journal code: 9206126. ISSN: 1018-8916.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199401
ENTRY DATE: Entered STN: 15 Feb 1994
Last Updated on STN: 29 Jan 1996
Entered Medline: 31 Jan 1994

AB Steel factor (S1F), also known as stem cell factor, is a potent growth stimulator of hemopoietic progenitor cells. In the context of transplantation of hemopoietic cells to irradiated allogeneic hosts, natural killer (NK) cells exert restrictive control on hemopoietic cell proliferation, and are regularly found in elevated concentration in areas of intense hemopoiesis. The present study was designed to examine the effects with time of S1F in vivo on the numbers of NK cells, identified by the presence of the NK 1.1 surface molecule, in the spleen and bone marrow. Throughout the first 3 days of S1F exposure, NK cell numbers, in spite of rapid (1 day) and significant increases in the other hemopoietic cell lineages, did not change in either the spleen or the bone marrow. However, NK cells were increased 2-fold in both organs by 7 days of S1F exposure. At this time, immature granuloid and erythroid cells and the large lymphoid cells in the spleen had more than doubled their respective control numbers and in the bone marrow, immature granuloid cells increased by 47% of control levels. The presence of a late, but not early, influence of S1F on NK cells of the spleen and bone marrow suggests an indirect effect of S1F on this lineage, occurring only when S1F-stimulated hemopoiesis becomes sufficiently intense, providing, thus, an abundance of NK cell targets.

L2 ANSWER 25 OF 103 MEDLINE on STN DUPLICATE 26
ACCESSION NUMBER: 91236532 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2032931
TITLE: Haematopoietic stem cell proliferation regulators investigated using an in vitro assay.
AUTHOR: Robinson S; Riches A

CORPORATE SOURCE: Department of Biology and Preclinical Medicine, University of St Andrews, Fife, UK.
SOURCE: Journal of anatomy, (1991 Feb) Vol. 174, pp. 153-62.
Journal code: 0137162. ISSN: 0021-8782.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199106
ENTRY DATE: Entered STN: 14 Jul 1991
Last Updated on STN: 14 Jul 1991
Entered Medline: 24 Jun 1991

AB The in vivo CFU-S murine haematopoietic transplantation assay has allowed haematopoietic stem cell behaviour and regulation to be investigated; however, the in vivo nature of the CFU-S assay restricts its use. Recent use of combinations of haematopoietic colony-stimulating factors (CSFs) in vitro has cloned a population of colony-forming cells from haematopoietic tissue, characterised by high proliferative potential and proposed to be a component of the haematopoietic stem cell compartment. A high proliferative potential colony-forming cell (HPP-CFC) population was assayed from murine haematopoietic tissue using a combination of WEHi 3B myelomonocytic leukaemic cell line conditioned medium (a crude source of interleukin 3 (IL3)/multi-CSF) and L929 fibroblast cell line conditioned medium (a crude source of M-CSF/CSF-1). The proportion of HPP-CFC in S-phase was determined following incubation with an S-phase specific, cytotoxic agent. In normal bone marrow from CBA/H mice, 9% of HPP-CFC were in S-phase, while in sublethally X-irradiated, regenerating bone marrow, 50% of HPP-CFC were in S-phase, a close correlation with in vivo CFU-S kinetics. The kinetic state of appropriate HPP-CFC populations can be modified in vitro by incubation with stem cell specific regulators (inhibitor and stimulator). Both inhibitor and stimulator were titratable against the appropriate target HPP-CFC population. Results obtained showed a close correlation between the in vivo CFU-S and in vitro HPP-CFC titration data, reinforcing the belief that the HPP-CFC population is a developmentally early haematopoietic precursor, possibly a component of the haematopoietic stem cell compartment.

L2 ANSWER 27 OF 103 MEDLINE on STN DUPLICATE 28
ACCESSION NUMBER: 90216404 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2323992
TITLE: Regulation of haematopoietic stem cell proliferation by stimulatory factors produced by murine fetal and adult liver.
AUTHOR: Dawood K A; Briscoe C V; Thomas D B; Riches A C
CORPORATE SOURCE: Department of Biology and Preclinical Medicine, University of St. Andrews, Scotland.
SOURCE: Journal of anatomy, (1990 Feb) Vol. 168, pp. 209-16.
Journal code: 0137162. ISSN: 0021-8782.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199005
ENTRY DATE: Entered STN: 22 Jun 1990
Last Updated on STN: 22 Jun 1990
Entered Medline: 24 May 1990

AB Haematopoietic stem cells in murine fetal liver are in a proliferative state unlike those in normal bone marrow which are quiescent. A regulatory activity is produced by cells in the fetal liver which will switch quiescent normal bone marrow haematopoietic stem cells into cell cycle in vitro. This regulator from Day 15 fetal liver cells is produced

by adherent cells and by cells fractionated on a Percoll gradient in the 1.064 and 1.076 g per cm3 density bands but not in the 1.123 g per cm3 band. Colony-stimulating factor cannot be detected in the supernatants containing the stem cell regulatory activity. The stimulator can be detected in supernatants produced from cell suspensions of liver cells at Day 15 and Day 17 of gestation and 24 hours and 72 hours after birth. However by 1 week after birth the production of the stimulator decreases and is undetectable 3 and 10 weeks after birth. The total numbers of haematopoietic stem cells (CFU-S) in fetal liver decrease from Day 15 of gestation and only small numbers are present 1 week after birth. Thus the decline in the production of haematopoietic stem cell proliferation stimulator correlates with the decrease in haematopoietic stem cell numbers in the liver through gestation and after birth.

L2 ANSWER 29 OF 103 MEDLINE on STN DUPLICATE 30
ACCESSION NUMBER: 88213448 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3259237
TITLE: The effects of recombinant CSF-1 on the blast cells of acute myeloblastic leukemia in suspension culture.
AUTHOR: Miyauchi J; Wang C; Kelleher C A; Wong G G; Clark S C; Minden M D; McCulloch E A
CORPORATE SOURCE: Ontario Cancer Institute, Toronto, Canada.
SOURCE: Journal of cellular physiology, (1988 Apr) Vol. 135, No. 1, pp. 55-62.
Journal code: 0050222. ISSN: 0021-9541.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198806
ENTRY DATE: Entered STN: 8 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 17 Jun 1988
AB Recombinant hemopoietic colony-stimulating factors (CSFs), including GM-CSF, G-CSF and IL-3, have been shown to be effective stimulators of both self-renewal and terminal differentiation of blast stem cells in acute myeloblastic leukemia (AML). We have examined the activity of a fourth growth factor, recombinant CSF-1 (or M-CSF), on the growth of leukemic blasts in culture. CSF-1 was found to be active on some, but not all, blast populations. In sensitive cells, CSF-1 often stimulated the production of adherent blast cells incapable of division. This observation leads us to suggest that CSF-1 may be useful in the treatment of selected cases of AML.

L2 ANSWER 30 OF 103 MEDLINE on STN DUPLICATE 31
ACCESSION NUMBER: 87313584 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3498096
TITLE: A stimulator of mouse stem cell proliferation produced by human regenerating bone marrow.
AUTHOR: Oishi H; Katsuno M; Umemura T; Nishimura J; Motomura S; Ibayashi H
SOURCE: Leukemia research, (1987) Vol. 11, No. 8, pp. 699-704.
Journal code: 7706787. ISSN: 0145-2126.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198710
ENTRY DATE: Entered STN: 5 Mar 1990
Last Updated on STN: 5 Mar 1990
Entered Medline: 19 Oct 1987
AB We examined CFU-S proliferation stimulator, which recruits

stem cells in DNA synthesis, in conditioned media prepared from bone marrow cells of patients with regeneration hemopoiesis after chemotherapy induced hypoplasia. This activity was estimated by hydroxyurea sensitivity of CFU-S in mice, under conditions of incubation with human bone marrow conditioned medium (BMCM). We found that CFU-S proliferation stimulator was present to a considerable extent in human regenerating BMCM, but less so in normal BMCM and that the production fluctuated with change of hemopoietic states, in the same patient. This stimulator was heat-labile, trypsin-sensitive and mainly produced by adherent cells. This factor may possibly be involved in regulation of proliferation of stem cells in regenerating bone marrow in humans.

L2 ANSWER 34 OF 103 MEDLINE on STN DUPLICATE 35
ACCESSION NUMBER: 87006212 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3530907
TITLE: Quantitative problems in bone marrow transplantation by peripheral blood stem cells.
AUTHOR: Serafimov-Dimitrov V
SOURCE: Haematologia, (1986) Vol. 19, No. 2, pp. 141-6.
Journal code: 0130266. ISSN: 0017-6559.
PUB. COUNTRY: Hungary
DOCUMENT TYPE: (CLINICAL TRIAL)
(COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198610
ENTRY DATE: Entered STN: 2 Mar 1990
Last Updated on STN: 2 Mar 1990
Entered Medline: 30 Oct 1986
AB Investigation into radiation bone marrow aplasia in mice, guinea pigs, dogs and clinical trials in man presented clear evidence of successful engraftment of autologous or allogeneic peripheral blood stem cells. The quantitative donation problems are discussed arising with the use of continuous cytopheresis to obtain a sufficient quantity of peripheral blood mononuclears (stem cells) for repopulation of aplastic bone marrow. Although bone marrow repopulation is possible by using peripheral blood mononuclears (stem cells) in individual cases, the method can only be used in practice after discovering an appropriate stimulator able to augment several times the number of bone marrow stem cells in the peripheral blood, or a new method for stem cell multiplication in vitro.

L2 ANSWER 43 OF 103 MEDLINE on STN DUPLICATE 44
ACCESSION NUMBER: 83061054 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7144230
TITLE: Stimulation of haemopoietic stem cell proliferation: characteristics of the stimulator -producing cells.
AUTHOR: Wright E G; Ali A M; Riches A C; Lord B I
SOURCE: Leukemia research, (1982) Vol. 6, No. 4, pp. 531-9.
Journal code: 7706787. ISSN: 0145-2126.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198301
ENTRY DATE: Entered STN: 17 Mar 1990
Last Updated on STN: 17 Mar 1990
Entered Medline: 27 Jan 1983
AB Media conditioned by regenerating murine bone marrow cells contain a stimulator of haemopoietic stem cell proliferation. Fractionated cell populations have been examined for

production of this stimulatory activity in order to characterize its cellular source. The stimulator is produced by adherent, phagocytic, radioresistant, Thy 1.2-, Fc+ cells in a population concentrated in a density range of 1.064-1.072 g/ml. The results indicate that the producer cells reside in the heterogenous mononuclear phagocytic population of the bone marrow.

L2 ANSWER 46 OF 103 MEDLINE on STN DUPLICATE 50
ACCESSION NUMBER: 81134497 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7470631
TITLE: Sources of haemopoietic stem cell proliferation: stimulators and inhibitors.
AUTHOR: Lord B I; Wright E G
SOURCE: Blood cells, (1980) Vol. 6, No. 4, pp. 581-93.
Journal code: 7513567. ISSN: 0340-4684.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198105
ENTRY DATE: Entered STN: 16 Mar 1990
Last Updated on STN: 16 Mar 1990
Entered Medline: 21 May 1981
AB Based on earlier findings that haemopoietic tissue contains extractable factors which are capable of specifically inhibiting or stimulating the movement of CFU-S into DNA synthesis, a series of preliminary experiments has now been carried out to investigate their cellular source(s), their activity in vivo, and their applicability to human problems. In vivo treatment of mice, in which femoral CFU-S are proliferating rapidly, with the inhibitory factor reduces the proportion of CFU-S in DNA synthesis to non-significant proportions. In addition, the inhibitor is capable of reducing the number of CFU-S induced to enter S following treatment with hydroxyurea, thus protecting CFU-S from the lethal effects of S-phase cytotoxic agents. Removal of specific types of marrow cells shows that both inhibitor and stimulator are adherent, phagocytic and, in the case of inhibitor, Thy--1-. These results suggest that the producer cells probably reside somewhere in the heterogeneous macrophage complex though their different densities suggest they are probably different cell types. Fresh human bone marrow is found to contain a very similar inhibitor and long-term cultures are also found to produce it continuously. The isolation of the producer cells may thus contribute to the understanding of normal physiological stem cell regulation and, by in vivo application, its eventual manipulation and protection.

L2 ANSWER 49 OF 103 MEDLINE on STN DUPLICATE 55
ACCESSION NUMBER: 79021839 MEDLINE
DOCUMENT NUMBER: PubMed ID: 308819
TITLE: Production of stem cell proliferation stimulators and inhibitors by haemopoietic cell suspensions.
AUTHOR: Wright E G; Lord B I
SOURCE: Biomedicine / [publiee pour l'A.A.I.C.I.G.], (1978 May-Jun) Vol. 28, No. 3, pp. 156-60.
Journal code: 0361342. ISSN: 0300-0893.
PUB. COUNTRY: France
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197812
ENTRY DATE: Entered STN: 14 Mar 1990
Last Updated on STN: 14 Mar 1990
Entered Medline: 27 Dec 1978
AB In mice treated with phenylhydrazine haemopoietic spleen colony forming

cells (CFU-S) are proliferating rapidly in the bone marrow but not in the spleen. Using such mice we have investigated the production of factors responsible for the control of CFU-S proliferation. When irradiated spleen cells are incubated with non-irradiated bone marrow cells there is a marked fall in the proportion of femoral CFU-S in DNA synthesis. In the converse experiments, rapid triggering of splenic CFU-S is achieved. Both these effects can be eliminated by washing the irradiated cells prior to incubation; they are, however, retained in the supernatant media "conditioned" by these cells. When the washed cells are incubated in fresh medium at 37 degrees C both stimulatory and inhibitory activities reappear but after different incubation periods. The data demonstrate that both proliferation stimulatory and inhibitory factors acting on CFU-S can be produced by the same haemopoietic cell suspension.

L2 ANSWER 51 OF 103 MEDLINE on STN DUPLICATE 57
ACCESSION NUMBER: 78000594 MEDLINE
DOCUMENT NUMBER: PubMed ID: 332243
TITLE: A stimulator of stem cell proliferation in regenerating bone marrow.
AUTHOR: Lord B I; Mori K J; Wright E G
SOURCE: Biomedicine / [publiee pour l'A.A.I.C.I.G.], (1977 Jul) Vol. 27, No. 6, pp. 223-6.
Journal code: 0361342. ISSN: 0300-0893.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197711
ENTRY DATE: Entered STN: 14 Mar 1990
Last Updated on STN: 14 Mar 1990
Entered Medline: 30 Nov 1977

AB A factor, capable of stimulating resting haemopoietic stem cells into DNA-synthesis has been extracted from regenerating bone marrow. It has a molecular weight in the range of 30.000-50.000 daltons and is not detectable in normal bone marrow. Used in combination with a stem cell proliferation inhibitor, previously described, it will restimulate proliferation in stem cells initially stopped by the inhibitor. Conversely, stimulation produced by this factor can be reversed by the addition of inhibitor. It is concluded that stem cell proliferation is controlled by an appropriate balance of endogenously produced stimulatory and inhibitory factors.

L2 ANSWER 58 OF 103 MEDLINE on STN
ACCESSION NUMBER: 74130076 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4150455
TITLE: Prostaglandin E2 as stimulator of haemopoietic stem cell proliferation.
AUTHOR: Feher I; Gidali J
SOURCE: Nature, (1974 Feb 22) Vol. 247, No. 442, pp. 550-1.
Journal code: 0410462. ISSN: 0028-0836.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197405
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 6 Feb 1995
Entered Medline: 28 May 1974

L2 ANSWER 60 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 45
ACCESSION NUMBER: 1982:86673 BIOSIS
DOCUMENT NUMBER: PREV198223016665; BR23:16665
TITLE: INTERFERON ITS ROLE IN RADIOPROTECTION AS A HEMATOPOIETIC

STEM CELL STIMULATOR.
AUTHOR(S): LVOVSKY E A [Reprint author]
CORPORATE SOURCE: DIV RADIAT ONCOL BIOPHYS, GEORGE WASHINGTON UNIV MED CENT, WASHINGTON, DC 20037, USA
SOURCE: International Journal of Radiation Oncology, Biology, Physics, (1981) Vol. 7, No. 9, pp. 1290-1291.
Meeting Info.: 23RD ANNUAL MEETING OF THE AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS, MIAMI BEACH, FLA., USA, OCT. 12-16, 1981. INT J RADIAT ONCOL BIOL PHYS.
CODEN: IOBPD3. ISSN: 0360-3016.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH

L2 ANSWER 66 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:140346 BIOSIS
DOCUMENT NUMBER: PREV200400133713
TITLE: Surgical injection of autologous, G-CSF mobilized, peripheral blood CD133+ cells for myocardial regeneration in patients undergoing coronary artery bypass grafting.
AUTHOR(S): Pompilio, Giulio [Reprint Author]; Cannata, Aldo [Reprint Author]; Capogrossi, Maurizio; Nascimbene, Angelo [Reprint Author]; Peccatori, Fedro; Biglioli, Paolo [Reprint Author]; Martinelli, Giovanni; Bertolini, Francesco
CORPORATE SOURCE: Cardiovascular Surgery and Gene-Cell Therapy, Cardiology "Monzino" Institute, Milan, Italy
SOURCE: Blood, (November 16 2003) Vol. 102, No. 11, pp. 335a. print.
Meeting Info.: 45th Annual Meeting of the American Society of Hematology. San Diego, CA, USA. December 06-09, 2003. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Mar 2004
Last Updated on STN: 10 Mar 2004

AB Bone-marrow stem cells are currently investigated as stimulators of myogenesis and angiogenesis in patients with a recent myocardial infarction, in candidates to coronary artery bypass grafting (CABG), or to induce angiogenesis in patients with refractory chronic angina not eligible for complete revascularization. Here we report a novel procedure for collection and surgical intramyocardial injection of peripheral blood stem cells (PBSC) in patients undergoing CABG. The protocol was approved by the Institutional Board and patients signed an informed consent. After study enrollment, 10 microg/Kg/d G-CSF were administered sc to the patient for 4-5 days to mobilize PBPC. Twelve-leads electrocardiogram was obtained daily. PBPC were collected on day 4-5 by 3-4 h apheresis, and CliniMacs was used to purify CD133+ cells. We collected 1-5X106 CD133+ cells/Kg (purity >90%, Viability >80%) in a final volume of 15-20 mL. CABG was scheduled for the day following apheresis to maintain cellular viability. The pericardium was opened and the myocardial regions target of PBSC injection recognized and inspected. After pre-load and after-load optimization, deep pericardial traction sutures were placed into the oblique sinus to obtain optimal exposure of the coronary vessels and myocardium. By cardiac wall stabilizer and endoluminar shunts, off-pump coronary bypass grafting was accomplished. PBSC were injected on a beating heart into the target myocardial areas by gentle hand injection. Needle covers were left in place and shortened (3 mm) to control PBPC injection (15-20 injections of 0.5-1.0 mL) into the myocardium. This constant depth avoided insufficient or excessive penetration. To induce myocardial repair, injections were accomplished along the border of the myocardial scar, directly visualized on the

beating-heart. Conversely, when cell therapy was conducted to generate angiogenesis, cells were delivered into the chronically ischemic ungraftable myocardium, identified by stress scintigraphy and 2-D ECG. We enrolled so far 4 patients. PBSC were intramyocardially delivered to repair a recent myocardial infarction (n=2) or injected in a large ischemic myocardial area not suitable for conventional revascularization (n=2). No cardiac or other complications were noted in early postoperative period or follow-up (3-9 m). In the two patients who underwent 5-m postoperative nuclear and angiographic reinvestigation, disappearance of both a previous inferior MI and a lateral wall ischemia were observed. Waiting for a longer follow-up in a larger series of patients, it is concluded that this novel approach of CABG and intramyocardial injection of blood-mobilized and purified CD133+ cells in a beating-heart is safe and feasible in patients with ischemic cardiomyopathy.

L2 ANSWER 71 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1996:256064 BIOSIS
DOCUMENT NUMBER: PREV199698812193
TITLE: An orally available small molecule stimulator of bone marrow stem cells accelerates postchemotherapy recovery of peripheral neutrophils and platelets.
AUTHOR(S): Morgan, A. S. [Reprint author]; Stanboli, A. [Reprint author]; Sanderson, P. [Reprint author]; Broxmeyer, H. E.
CORPORATE SOURCE: Terrapin Technologies, South San Francisco, CA 94080, USA
SOURCE: Proceedings of the American Association for Cancer Research Annual Meeting, (1996) Vol. 37, No. 0, pp. 288.
Meeting Info.: 87th Annual Meeting of the American Association for Cancer Research. Washington, D.C., USA. April 20-24, 1996.
ISSN: 0197-016X.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 31 May 1996
Last Updated on STN: 31 May 1996

L2 ANSWER 79 OF 103 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1979:127045 BIOSIS
DOCUMENT NUMBER: PREV197967007045; BA67:7045
TITLE: EXPERIMENTAL AND CLINICAL INVESTIGATIONS ON STEM CELL TAKE AND COLONY FORMATION.
AUTHOR(S): ASTALDI G [Reprint author]; BAGNARA G P; BRUNELLI M A; KARANOVIC D; KARANOVIC J; SCORZA R; TOPUZ U
CORPORATE SOURCE: BLOOD RES FOUND CENT, HOSP TORTONA, 15057 TORTONA, ITALY
SOURCE: Haematologia, (1977) Vol. 11, No. 1-2, pp. 11-30.
CODEN: HAEMBY. ISSN: 0017-6559.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB Lymphocyte transplantation into total body-irradiated rats was discussed. The effect on spleen colony formation caused by the transplantation of untreated lymphocytes, as well as of lymphocytes previously incubated with PHA [phytohemagglutinin], with PHA plus L-asparaginase, or with lymphokines was studied. The effect of the urinary colony-stimulating factor in vitro, and the in vitro feeder-layer activity of leukocytes on colony formation of human and mice bone marrow cells in hematological diseases was discussed. The injection of rat lymphocytes previously incubated for 24 h with PHA resulted in a higher number and a larger size of colonies in the spleen of the recipient rats. Lymphocytes preincubated with lymphokines gave rise to the formation of spleen colonies which were larger than those developing after the injection of untreated lymphocytes.

When the lymphocytes were previously incubated with PHA plus L-asparaginase, PHA failed to stimulate colony formation in the spleen. The phenomenon is explained by assuming that PHA, as an aspecific stimulator of cell division, initiated the division of CFUs [pluripotential stem cells]. The CFUs content of the preincubated samples increased, resulting in an increase in the number of colonies formed after the transplantation of lymphocytes pretreated with PHA. Another possible explanation is that CFUs division, or their spleen take was enhanced by the immunocompetent lymphocytes activated by PHA, either directly or via soluble mediators produced or released by immunocompetent lymphocytes such as lymphokines. The study of colony-forming cells and colony-stimulating activity in primary myelofibrosis (PM) showed an increase in the number of circulating CFUc [granulocyte progenitor cells] in this conditions, and an abnormal density of these cells reaching a peak below 1.062. The lowering of CSA [colony stimulating activity] in the first 2 peripheral blood gradient fractions agreed with the observation in the same fractions ofa high percentage of CFUc at the expense of the CSC population. Double cell population seems to exist in PM. One is greatly abnormal with a low specific density and high plating efficiency; the other population is almost normal, showing a higher specific density and a lower plating efficiency.

L2 ANSWER 82 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:716893 CAPLUS
DOCUMENT NUMBER: 141:218997
TITLE: Inhibitor and stimulator of stem cell proliferation and uses thereof
INVENTOR(S): Wolpe, Stephen D.; Tsyrlova, Irena
PATENT ASSIGNEE(S): Wellstat Therapeutics Corporation, USA
SOURCE: U.S., 70 pp., Cont.-in-part of U.S. Ser. No. 627,173.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6784155	B1	20040831	US 1997-832443	19970403
US 5861483	A	19990119	US 1996-627173	19960403 <--
CA 2249716	A1	19971009	CA 1997-2249716	19970403 <--
CN 1220670	A	19990623	CN 1997-195095	19970403 <--
CN 1541706	A	20041103	CN 2004-10007470	19970403
ES 2252781	T3	20060516	ES 1997-920117	19970403
EP 1820805	A2	20070822	EP 2005-19956	19970403
R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, AL, LT, LV, RO, SI				
ZA 9802746	A	19990329	ZA 1998-2746	19980401 <--
KR 2000005428	A	20000125	KR 1998-708185	19981002 <--
NZ 504512	A	20011026	NZ 2000-504512	20000512 <--
AU 768081	B2	20031204	AU 2001-23195	20010223 <--
US 2004167060	A1	20040826	US 2004-776172	20040212
US 7115267	B2	20061003		
HK 1069985	A1	20070309	HK 2005-102673	20050330
US 2006166863	A1	20060727	US 2006-386736	20060323

PRIORITY APPLN. INFO.:		
US 1996-627173	A2	19960403
AU 1997-24391	A3	19970403
EP 1997-920117	A3	19970403
NZ 1997-331895	A1	19970403
US 1997-832443	A	19970403
WO 1997-US5601	W	19970403
US 2004-776172	A3	20040212

AB Disclosed and claimed are methods for the isolation and use of stem cell modulating factors for regulating stem cell cycle and for accelerating the post-chemotherapy peripheral blood cell recovery. Also disclosed and

claimed are the inhibitors and stimulators of stem cell proliferation. Hb α-chain fragments are described that have the desired properties.

REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 95 OF 103 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1996:684559 CAPLUS
DOCUMENT NUMBER: 126:6155
TITLE: Purification and identification of a hematopoietic stem cell proliferation stimulator from human fetal liver
AUTHOR(S): Wen, Geng-Yun; Wu, Zu-Ze; He, Fu-Chu; Pei, Xue-Tao
CORPORATE SOURCE: Inst. Radiation med., Beijing, 100850, Peop. Rep. China
SOURCE: Shengwu Huaxue Zazhi (1996), 12(5), 569-573
CODEN: SHZAE4; ISSN: 1000-8543
PUBLISHER: Zhongguo Shengwu Huaxue Yu Fenzi Shengwu Xuehui
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB Through several steps including ultrafiltration, chromatog. on DEAE-Sephacel, Sephacryl S-200 and HPLC-Superose 12 HR, a substance of 35 kD termed as FLS-4 was isolated from human fetal livers of 3-5 mo with highly activity of stimulating hematopoietic stem cell proliferation. In phys. and biol. nature, FLS-4 exhibited a unique character different from IL-3, IL-6, CM-CSF and FLT3 ligand which are known to have hematopoietic stem cell proliferation stimulating activity of different extent. FLS-4 is very likely to be a novel hematopoietic stem cell proliferation stimulator. During the period of active hematopoiesis in fetal liver, FLS-4 might be the major candidate in triggering hematopoietic stem cells from resting G0 into cytocycling phase.

L2 ANSWER 100 OF 103 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1982145728 EMBASE
TITLE: Regulation of pluripotent stem cell proliferation and differentiation: The role of long-range humoral factors.
AUTHOR: Tubiana M.; Frindel E.
CORPORATE SOURCE: Inst. Radiobiol., Clin., Inst. Gustave-Roussy, 94800 Villejuif, France
SOURCE: Journal of Cellular Physiology, (1982) Vol. 110, No. Suppl. 1, pp. 13-21.
ISSN: 0021-9541 CODEN: JCLLAX
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 029 Clinical and Experimental Biochemistry
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991
AB The proliferative status of the hemopoietic pluripotential stem cells (CFU-S) is controlled by inhibitors and stimulators , which have been studied by an in vivo-in vitro technique. Inhibitors protect CFU-S during iterative administration of cycle specific drugs. Among stimulators are long-range humoral factors that are released by bone marrow following irradiation or drug administration. After the same treatment, bone marrow also releases a long-range humoral factor that increases the rate of differentiation of CFU-S, probably in order to compensate for the depletion of the maturing compartment. This differentiation is qualitatively different from normal differentiation. When the bone marrow of mice treated with Ara-C is transplanted to lethally irradiated mice, the total number of nodules remains constant; however the number of erythroid (E) colonies in the spleen is significantly increased, while the number of granulocytic (G) colonies is significantly decreased, and the number of mixed colonies is slightly

decreased. Similar observations for E and G colonies are made when normal bone marrow is injected into lethally irradiated mice following in vitro incubation with humoral factors released by cytosine-arabinoside (Ara-C) treated mice. In both cases most splenic colonies contain CFU-S and GM-CFC, even when they appear histologically E colonies. After irradiation or iterative administration of Ara-C the E/G ratio is decreased. The factors involved, pluripoietins, are released by both bone marrow and spleen and are found in the serum of treated mice. The mechanism by which they act is unknown; however two hypotheses can be made: a) Modulation of differentiation potential towards only one of the cell lineages. During 6 to 7 days after ARa-C administration the determination of CFU-S is modified. However this restriction to erythroid determination is temporary. This hypothesis is compatible with the 'hemopoietic inductive model', but microenvironment is not playing any role. b) Specific inhibition or stimulation of the committed stem cells to which the CFU-S having received the 'message' gives birth. This information lasts during one week but disappears when the cells are plated in vitro. Whatever the mechanism, the primary events occur at CFU-S level and the information is transmitted to the descendants, showing that humoral factors can channel the differentiation pathways.

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AT 12:51:12 ON 24 SEP 2007
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FILE 'BIOSIS' ENTERED AT 12:51:12 ON 24 SEP 2007
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CA SUBSCRIBER PRICE	-2.34	-2.34

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     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 10:32:53 ON 24 SEP 2007
L1   220 S (STEM(A)CELL)(S)(STIMULATOR OR DIFFERENTIATOR) AND PD<=200404
L2   103 DUP REM L1 (117 DUPLICATES REMOVED)
L3   2 S L2 AND (BETA(A)CELL OR LANGERHAN?)

=> S (STEM(A)CELL)(S)(Recruiting(3A)factor) AND pd<=20040415

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   2 FILES SEARCHED...
L4          16 (STEM(A) CELL)(S)(RECRUITING(3A) FACTOR) AND PD<=20040415

=> Dup rem l4
PROCESSING COMPLETED FOR L4
L5             7 DUP REM L4 (9 DUPLICATES REMOVED)
                 ANSWERS '1-5' FROM FILE MEDLINE
                 ANSWERS '6-7' FROM FILE BIOSIS

=> D Ibib abs L5 1-7

L5   ANSWER 1 OF 7     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:   2003286492     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 12799282
TITLE:              Angiogenic factors reconstitute hematopoiesis by
                    recruiting stem cells from bone
                    marrow microenvironment.
AUTHOR:             Rafii Shahin; Avecilla Scott; Shmelkov Sergey; Shido Koji;
                    Tejada Rafael; Moore Malcolm A S; Heissig Beate; Hattori
                    Koichi
CORPORATE SOURCE:   Cornell University Medical College, New York, New York
                    10021, USA.. sraffi@med.cornell.edu
SOURCE:             Annals of the New York Academy of Sciences, (2003
                    May) Vol. 996, pp. 49-60.
                    Journal code: 7506858. ISSN: 0077-8923.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200307
ENTRY DATE:         Entered STN: 20 Jun 2003
                    Last Updated on STN: 25 Jul 2003
                    Entered Medline: 24 Jul 2003
AB   The mechanism by which angiogenic factors recruit bone marrow (BM)-derived
     quiescent endothelial and hematopoietic stem cells (HSCs) is not known.
     Here, we report that functional vascular endothelial growth factor
     receptor-1 (VEGFR1, Flt-1) is expressed on a subpopulation of human
     CD34(+) and mouse Lin-Sca-1(+)c-Kit(+) BM-repopulating stem cells,
     conveying signals for recruitment of HSCs and reconstitution of
     hematopoiesis.  Inhibition of VEGFR1 signaling, but not VEGFR2 (Flk-1,
     KDR), blocked HSC cell cycling, differentiation and hematopoietic recovery
     after BM suppression, resulting in the demise of the treated mice.  Plasma
     elevation of placental growth factor (PlGF), which signals through VEGFR1,
     but not VEGFR2, restored hematopoiesis during the early and late phases
     following BM suppression.  The mechanism whereby PlGF enhanced early
     phases of BM recovery was mediated directly through rapid chemotaxis of
     readily available VEGFR1(+) BM-repopulating and progenitor cells.  The
     late phase of hematopoietic recovery was driven by PlGF-induced
     upregulation of matrix metalloproteinase-9 (MMP-9) in the BM, mediating
     the release of soluble Kit-ligand (sKitL). sKitL increased proliferation
     and motility of HSCs and progenitor cells, thereby augmenting
     hematopoietic recovery.  PlGF promotes recruitment of VEGFR1(+) HSCs from
     a quiescent to a proliferative microenvironment within the BM, favoring
     differentiation, mobilization, and reconstitution of hematopoiesis.

L5   ANSWER 2 OF 7     MEDLINE on STN                    DUPLICATE 2
ACCESSION NUMBER:   2002402553     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 12091880
TITLE:              Placental growth factor reconstitutes
                    hematopoiesis by recruiting VEGFR1(+)
                    stem cells from bone-marrow
                    microenvironment.
AUTHOR:             Hattori Koichi; Heissig Beate; Wu Yan; Dias Sergio; Tejada
                    Rafael; Ferris Barbara; Hicklin Daniel J; Zhu Zhenping;
                    Bohlen Peter; Witte Larry; Hendrikx Jan; Hackett Neil R;
```

Crystal Ronald G; Moore Malcolm A S; Werb Zena; Lyden David; Rafii Shahin

CORPORATE SOURCE: Department of Medicine, Cornell University Medical College, New York, New York, USA.

CONTRACT NUMBER: AR46238 (NIAMS)
CA 72006 (NCI)
CA 75072 (NCI)
NS39278 (NINDS)
R01 HL-58707 (NHLBI)
R01 HL-66592 (NHLBI)
R01 HL-67839 (NHLBI)
R01 HL61401 (NHLBI)
R01 HL61849 (NHLBI)

SOURCE: Nature medicine, (2002 Aug) Vol. 8, No. 8, pp. 841-9. Electronic Publication: 2002-07-01.
Journal code: 9502015. ISSN: 1078-8956.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200209

ENTRY DATE: Entered STN: 2 Aug 2002
Last Updated on STN: 7 Sep 2002
Entered Medline: 6 Sep 2002

AB The mechanism by which angiogenic factors recruit bone marrow (BM)-derived quiescent endothelial and hematopoietic stem cells (HSCs) is not known. Here, we report that functional vascular endothelial growth factor receptor-1 (VEGFR1) is expressed on human CD34(+) and mouse Lin(-)Sca-1(+)c-Kit(+) BM-repopulating stem cells, conveying signals for recruitment of HSCs and reconstitution of hematopoiesis. Inhibition of VEGFR1, but not VEGFR2, blocked HSC cell cycling, differentiation and hematopoietic recovery after BM suppression, resulting in the demise of the treated mice. Placental growth factor (PlGF), which signals through VEGFR1, restored early and late phases of hematopoiesis following BM suppression. PlGF enhanced early phases of BM recovery directly through rapid chemotaxis of VEGFR1(+) BM-repopulating and progenitor cells. The late phase of hematopoietic recovery was driven by PlGF-induced upregulation of matrix metalloproteinase-9, mediating the release of soluble Kit ligand. Thus, PlGF promotes recruitment of VEGFR1(+) HSCs from a quiescent to a proliferative BM microenvironment, favoring differentiation, mobilization and reconstitution of hematopoiesis.

L5 ANSWER 3 OF 7 MEDLINE on STN DUPLICATE 3

ACCESSION NUMBER: 95106812 MEDLINE

DOCUMENT NUMBER: PubMed ID: 7528861

TITLE: Primitive multilineage progenitor cells predominate in peripheral blood early after mobilization with high-dose cyclophosphamide and GM-CSF or G-CSF.

AUTHOR: Croockewit S; Raymakers R; Trilsbeek C; Dolstra H; Pennings A; de Witte T

CORPORATE SOURCE: Division of Hematology, University Hospital Nijmegen, The Netherlands.

SOURCE: Leukemia : official journal of the Leukemia Society of America, Leukemia Research Fund, U.K, (1994 Dec) Vol. 8, No. 12, pp. 2194-9.
Journal code: 8704895. ISSN: 0887-6924.

PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199501

ENTRY DATE: Entered STN: 15 Feb 1995

Last Updated on STN: 29 Jan 1996
Entered Medline: 27 Jan 1995

AB The change in phenotype, number and proliferative capacity of peripheral blood hematopoietic progenitors (PBHP) was studied in six patients with multiple myeloma during hematopoietic recovery after mobilization with high-dose cyclophosphamide and GM-CSF or G-CSF. In all six patients the first CD34+ cells appearing in the peripheral blood (PB) after cytoreductive treatment were predominantly CD34+/33- (> 70%). At later stages when leukapheresis procedures were started, the CD34+/33+ cells predominated in five of six patients. In leukapheresis harvests of peripheral blood, and in bone marrow addition of SCF and IL-6 to the culturing medium enhanced the plating efficiency. In peripheral blood an increase from 12 to 22% for CD34+/33+ and from 6 to 14% for CD34+/33- was observed. In normal bone marrow we observed an increase from 15 to 23% for CD34+/33+ and from 7 to 17% for CD34+/33-. Highly proliferative progenitors (>500 cells) in the CD34+/33- fraction appeared to be dependent on the addition of 'stem cell recruiting factors' (SCF and IL-6); in bone marrow the percentage of wells with >500 cells increased from 0.9 to 12.6% after SCF+IL-6 and in PBHP from 2 to 9%. We conclude that the first progenitors appearing in the peripheral blood after priming with high-dose cyclophosphamide and GM- or G-CSF have a more primitive immunophenotype, CD34+/33-.

L5 ANSWER 4 OF 7 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 94284025 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7516921
TITLE: Peripheral blood cell harvests yield primitive multilineage progenitor cells in the CD34+/33- fraction.
AUTHOR: Croockewit A; Raymakers R A; Trilsbeek C; Dolstra H; Pennings A; De Witte T J; Haanen C
CORPORATE SOURCE: Division of Hematology, University Hospital Nijmegen, The Netherlands.
SOURCE: The International journal of artificial organs, (1993 Dec) Vol. 16 Suppl 5, pp. 83-8.
Journal code: 7802649. ISSN: 0391-3988.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199407
ENTRY DATE: Entered STN: 10 Aug 1994
Last Updated on STN: 29 Jan 1996
Entered Medline: 25 Jul 1994

AB The presence of primitive hematopoietic progenitor cells or stem cells in peripheral blood (PBSC's) harvests was investigated in a single cell culturing assay and compared with the results obtained in aspirates of normal bone marrow. Based on the presence of CD33, rather differentiated progenitor cells (CD34+/33+) were distinguished from more primitive cells (CD34+/33-). The growth potential of CD34+/33+ and CD34+/33- cells have been studied. Single cell sorting was performed from peripheral blood harvests, obtained from three patients with multiple myeloma during hematopoietic recovery after treatment with high dose cyclophosphamide and rhu-GM-CSF. To test the effect of "stem cell recruiting factors" the cells were sorted in 96-well plates, prefilled with liquid medium both in the presence of IL-3 + G-CSF+GM-CSF+Epo and the same growth factors supplemented with SCF+IL-6. Addition of SCF and IL-6 to the culturing medium enhanced the plating efficiency of CD34+/33- cells considerably more than that of CD34+/33+ cells. This was observed in harvests of peripheral blood as well as in aspirates of normal bone marrow. The differences between CD34+/33+ and CD34+/33- were even more pronounced when only the large colonies (> 500 cells/well) were taken into consideration. Assuming that IL-6 and SCF are "stem cell recruiting factors,"

the CD34+/33- fraction contains more clonogenic cells than the CD34+/33+ fraction. In all three patients the first CD34+ cells appearing in the peripheral blood (PB) after cytoreductive treatment were predominantly CD34+/33- (> 80%). At later stages when the leukocyte counts had reached higher values the CD34+/33+ cells predominated. (ABSTRACT TRUNCATED AT 250 WORDS)

L5 ANSWER 5 OF 7 MEDLINE on STN
ACCESSION NUMBER: 90364927 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2203234
TITLE: [Interleukin 1 and hematopoiesis].
Interleukin 1 und Hamatopoese.
AUTHOR: Schaffner H
CORPORATE SOURCE: Wissenschaftsbereich Tierphysiologie der Sektion Biowissenschaften, Karl-Marx-Universitat Leipzig.
SOURCE: Allergie und Immunologie, (1990) Vol. 36, No. 2, pp. 77-86. Ref: 54
Journal code: 0314702. ISSN: 0323-4398.
PUB. COUNTRY: GERMANY, EAST: German Democratic Republic
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199010
ENTRY DATE: Entered STN: 9 Nov 1990
Last Updated on STN: 9 Nov 1990
Entered Medline: 2 Oct 1990
AB Interleukin-1 mediates a broad spectrum of activities in the functional network of cytokines. In addition to its function as an inducer of the acute phase response IL-1 has many effects on hemopoiesis in normal and hematologically impaired organisms. This regulatory function is realized by its ability to stimulate the release of hematopoietic growth factors and by its recruiting property for cell cycles of different hemopoietic progenitors and stem cells. IL-1 acts synergistically with the colony-stimulating factors.

L5 ANSWER 6 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:351870 BIOSIS
DOCUMENT NUMBER: PREV200300351870
TITLE: Angiogenic factors reconstitute hematopoiesis by recruiting stem cells from bone marrow microenvironment.
AUTHOR(S): Rafii, Shahin [Reprint Author]; Avecilla, Scott; Shmelkov, Sergey; Shido, Koji; Tejada, Rafael; Moore, Malcolm A. S.; Heissig, Beate; Hattori, Koichi
CORPORATE SOURCE: Division of Hematology-Oncology, Cornell University Medical College, 1300 York Avenue, Room D601, New York, NY, 10021, USA
srafii@med.cornell.edu
SOURCE: Orlic, Donald [Editor, Reprint Author]; Bruemmendorf, Tim H. [Editor]; Fibbe, Willem [Editor]; Sharkis, Saul [Editor]; Kanz, Lothan [Editor]. (2003) pp. 49-60. Hematopoietic stem cells 2002: Genetics and function. print.
Publisher: New York Academy of Sciences, 2 East 63rd Street, New York, NY, 10021, USA. Series: Annals of the New York Academy of Sciences.
Meeting Info.: Fourth International Symposium on Hematopoietic Stem Cells. Tuebingen, Germany. September 19-21, 2002.
ISSN: 0077-8923 (ISSN print). ISBN: 1-57331-466-8 (cloth).
DOCUMENT TYPE: Book; (Book Chapter)
Conference; (Meeting)
Conference; (Meeting Paper)

LANGUAGE: English
ENTRY DATE: Entered STN: 30 Jul 2003
Last Updated on STN: 30 Jul 2003

L5 ANSWER 7 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1982:223242 BIOSIS
DOCUMENT NUMBER: PREV198273083226; BA73:83226
TITLE: REGULATION OF MACROPHAGE POPULATION 3. THE IMMUNOLOGIC INDUCTION OF EXUDATES RICH IN IA BEARING MACROPHAGES IS A RADIO SENSITIVE PROCESS.
AUTHOR(S): SCHER M G [Reprint author]; UNANUE E R; BELLER D I
CORPORATE SOURCE: DEP OF PATHOLOGY, HARVARD MEDICAL SCH, BOSTON, MASS 02115, USA
SOURCE: Journal of Immunology, (1982) Vol. 128, No. 1, pp. 447-450.
CODEN: JOIMA3. ISSN: 0022-1767.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
AB Some of the conditions regulating the selective augmentation of the Ia-positive macrophage population within immunologically induced exudates were evaluated. Antigen-stimulated T cells secrete a protein referred to as macrophage-(Ia-positive) recruiting factor (MIRF), which when injected i.p. stimulates a 10- to 20-fold increase in the number of Ia-positive exudate macrophages. This response is totally abrogated when mice are lethally irradiated before injection of MIRF or immune T cells. Adoptive transfer of bone marrow cells to irradiated mice substantially restores their ability to respond to the immunologic stimuli, even if the transferred bone marrow was itself depleted of Ia-positive cells. The high level of Ia-positive macrophages induced by MIRF apparently requires a renewable stem cell source in order to be maintained. Even when macrophages were elicited by injecting thioglycollate before irradiation, Ia-positive cells were not induced in response to MIRF. The target of MIRF in vivo may be restricted to a developmentally young cell within or recently derived from a stem cell compartment such as the bone marrow and Ia-positive and Ia-negative macrophages ultimately derive from a potentially common Ia-negative stem cell.

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     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 10:32:53 ON 24 SEP 2007
L1          220 S (STEM(A)CELL)(S)(STIMULATOR OR DIFFERENTIATOR) AND PD<=200404
L2          103 DUP REM L1 (117 DUPLICATES REMOVED)
L3            2 S L2 AND (BETA(A)CELL OR LANGERHAN?)
L4           16 S (STEM(A)CELL)(S)(RECRUITING(3A)FACTOR) AND PD<=20040415
L5            7 DUP REM L4 (9 DUPLICATES REMOVED)

=> S (BETA(A)CELL OR LANGERHAN?) (L)(EPO OR GM-CSF OR SCF OR G-CSF) AND pd<=20040415
   2 FILES SEARCHED...
L6          844 (BETA(A) CELL OR LANGERHAN?) (L)(EPO OR GM-CSF OR SCF OR G-CSF)
              AND PD<=20040415

=> S (BETA(A)CELL OR LANGERHAN?)(S)(EPO OR GM-CSF OR SCF OR G-CSF) AND pd<=20040415
   1 FILES SEARCHED...
L7          300 (BETA(A) CELL OR LANGERHAN?)(S)(EPO OR GM-CSF OR SCF OR G-CSF)
              AND PD<=20040415

=> S ((BETA(A)CELL OR LANGERHAN?)(S)regeneration) (S)(EPO OR GM-CSF OR SCF OR
G-CSF) AND pd<=20040415
   2 FILES SEARCHED...
L8            0 ((BETA(A) CELL OR LANGERHAN?)(S) REGENERATION) (S)(EPO OR GM-CSF
                OR SCF OR G-CSF) AND PD<=20040415

=> Dup rem L7
PROCESSING COMPLETED FOR L7
L9          147 DUP REM L7 (153 DUPLICATES REMOVED)
                ANSWERS '1-69' FROM FILE MEDLINE
                ANSWERS '70-94' FROM FILE BIOSIS
                ANSWERS '95-141' FROM FILE CAPLUS
                ANSWERS '142-147' FROM FILE EMBASE

=> D Ti L9 1-69

L9   ANSWER 1 OF 147     MEDLINE on STN                  DUPLICATE 1
TI   Expression of milk fat globule epidermal growth factor 8 in immature
     dendritic cells for engulfment of apoptotic cells.

L9   ANSWER 2 OF 147     MEDLINE on STN                  DUPLICATE 2
TI   Increased islet antigen presentation leads to type-1 diabetes in mice with
     autoimmune susceptibility.

L9   ANSWER 3 OF 147     MEDLINE on STN                  DUPLICATE 3
TI   XPA gene-deficient, SCF-transgenic mice with epidermal melanin are
     resistant to UV-induced carcinogenesis.

L9   ANSWER 4 OF 147     MEDLINE on STN                  DUPLICATE 4
TI   Interleukin-3 in cooperation with transforming growth factor beta induces
     granulocyte macrophage colony stimulating factor independent
     differentiation of human CD34+ hematopoietic progenitor cells into
     dendritic cells with features of Langerhans cells.

L9   ANSWER 5 OF 147     MEDLINE on STN                  DUPLICATE 6
TI   Down-regulation of Toll-like receptor expression in monocyte-derived
     Langerhans cell-like cells: implications of low-responsiveness to
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bacterial components in the epidermal Langerhans cells.

L9 ANSWER 6 OF 147 MEDLINE on STN DUPLICATE 7
TI Withdrawal of TNF-alpha after the fifth day of differentiation of CD34+ cord blood progenitors generates a homogeneous population of Langerhans cells and delays their maturation.

L9 ANSWER 7 OF 147 MEDLINE on STN DUPLICATE 8
TI Novel membrane-bound GM-CSF vaccines for the treatment of cancer: generation and evaluation of mbGM-CSF mouse B16F10 melanoma cell vaccine.

L9 ANSWER 8 OF 147 MEDLINE on STN DUPLICATE 9
TI TGF-beta 1 synergizes with GM-CSF to promote the generation of glial cell-derived dendriform cells in vitro.

L9 ANSWER 9 OF 147 MEDLINE on STN DUPLICATE 11
TI Immune responses to tumour antigens: implications for antigen specific immunotherapy of cancer.

L9 ANSWER 10 OF 147 MEDLINE on STN DUPLICATE 12
TI Control of the differentiation state and function of human epidermal Langerhans cells by cytokines in vitro.

L9 ANSWER 11 OF 147 MEDLINE on STN DUPLICATE 13
TI [The Langerhans cell: from in vitro production to use in cellular immunotherapy].
La cellule de Langerhans: de la production in vitro a l'utilisation en immunotherapie cellulaire.

L9 ANSWER 12 OF 147 MEDLINE on STN DUPLICATE 14
TI Langerhans cells differentiation: a three-act play.

L9 ANSWER 13 OF 147 MEDLINE on STN DUPLICATE 15
TI Large-scale culture and selective maturation of human Langerhans cells from granulocyte colony-stimulating factor-mobilized CD34+ progenitors.

L9 ANSWER 14 OF 147 MEDLINE on STN DUPLICATE 16
TI Effect of granulocyte-macrophage colony-stimulating factor on the generation of epidermal Langerhans cells.

L9 ANSWER 15 OF 147 MEDLINE on STN DUPLICATE 17
TI Intradermal granulocyte-macrophage colony-stimulating factor alters cutaneous antigen-presenting cells and differentially affects local versus distant immunization in humans.

L9 ANSWER 16 OF 147 MEDLINE on STN DUPLICATE 18
TI Tumor cell surface expression of granulocyte-macrophage colony-stimulating factor elicits antitumor immunity and protects from tumor challenge in the P815 mouse mastocytoma tumor model.

L9 ANSWER 17 OF 147 MEDLINE on STN DUPLICATE 19
TI Transforming growth factor-beta1 polarizes murine hematopoietic progenitor cells to generate Langerhans cell-like dendritic cells through a monocyte/macrophage differentiation pathway.

L9 ANSWER 18 OF 147 MEDLINE on STN DUPLICATE 20
TI Differential effects of cytokines and immunosuppressive drugs on CD40, B7-1, and B7-2 expression on purified epidermal Langerhans cells1.

L9 ANSWER 19 OF 147 MEDLINE on STN DUPLICATE 21
TI IL-4 inhibits the migration of human Langerhans cells through the downregulation of TNF receptor II expression.

L9 ANSWER 20 OF 147 MEDLINE on STN DUPLICATE 22
TI Human dendritic cells express a 95 kDa activation/differentiation antigen

defined by CMRF-56.

L9 ANSWER 21 OF 147 MEDLINE on STN DUPLICATE 23
TI Injection of DNA encoding granulocyte-macrophage colony-stimulating factor recruits dendritic cells for immune adjuvant effects.

L9 ANSWER 22 OF 147 MEDLINE on STN DUPLICATE 25
TI Expression of maturation-/migration-related molecules on human dendritic cells from blood and skin.

L9 ANSWER 23 OF 147 MEDLINE on STN DUPLICATE 26
TI IL-4 addition during differentiation of CD34 progenitors delays maturation of dendritic cells while promoting their survival.

L9 ANSWER 24 OF 147 MEDLINE on STN DUPLICATE 27
TI Effect of granulocyte macrophage-colony stimulating factor on Langerhans cells in normal and healthy atopic subjects.

L9 ANSWER 25 OF 147 MEDLINE on STN DUPLICATE 28
TI GM-CSF promotes differentiation of a precursor cell of monocytes and Langerhans-type dendritic cells from CD34+ haemopoietic progenitor cells.

L9 ANSWER 26 OF 147 MEDLINE on STN DUPLICATE 29
TI Characterisation of two human dendritic cell-lines that express CD1a, take-up, process and present soluble antigens and induce MLR.

L9 ANSWER 27 OF 147 MEDLINE on STN DUPLICATE 30
TI Genetic modification of a carcinoma with the IL-4 gene increases the influx of dendritic cells relative to other cytokines.

L9 ANSWER 28 OF 147 MEDLINE on STN DUPLICATE 31
TI Productive infection of dendritic cells by HIV-1 and their ability to capture virus are mediated through separate pathways.

L9 ANSWER 29 OF 147 MEDLINE on STN DUPLICATE 32
TI DNA polymorphisms and mutations of the tumor necrosis factor-alpha (TNF-alpha) promoter in Langerhans cell histiocytosis (LCH).

L9 ANSWER 30 OF 147 MEDLINE on STN DUPLICATE 33
TI CD34+ peripheral blood progenitor cell and monocyte derived dendritic cells: a comparative analysis.

L9 ANSWER 31 OF 147 MEDLINE on STN DUPLICATE 34
TI Alteration of the CD34+ Tf-1 beta cell line profile in response to long-term exposure to IL-15.

L9 ANSWER 32 OF 147 MEDLINE on STN DUPLICATE 35
TI Modulation of MHC class II+ Langerhans cell numbers in corticosteroid treated epidermis by GM-CSF in combination with TNF-alpha.

L9 ANSWER 33 OF 147 MEDLINE on STN DUPLICATE 36
TI Interleukin-3 cooperates with tumor necrosis factor alpha for the development of human dendritic/Langerhans cells from cord blood CD34+ hematopoietic progenitor cells.

L9 ANSWER 34 OF 147 MEDLINE on STN DUPLICATE 37
TI Role of granulocyte-macrophage colony stimulating factor (GM-CSF) in the pathogenesis of adult pulmonary histiocytosis X.

L9 ANSWER 35 OF 147 MEDLINE on STN DUPLICATE 38
TI Interleukin-1 beta and granulocyte-macrophage colony-stimulating factor mediate Langerhans cell maturation differently.

L9 ANSWER 36 OF 147 MEDLINE on STN DUPLICATE 40
TI Flow cytometric analysis of cytokine receptors on human Langerhans' cells. Changes observed after short-term culture.

L9 ANSWER 37 OF 147 MEDLINE on STN DUPLICATE 41
TI In vitro HIV1 infection of CD34+ progenitor-derived dendritic/ Langerhans cells at different stages of their differentiation in the presence of GM-CSF/TNF alpha.

L9 ANSWER 38 OF 147 MEDLINE on STN DUPLICATE 42
TI Macrophage colony-stimulating factor (M-CSF) inhibits the decrease in the amount of rRNA and IA beta mRNA in cultured epidermal Langerhans cells of the mouse.

L9 ANSWER 39 OF 147 MEDLINE on STN DUPLICATE 43
TI Selected strategies to augment polynucleotide immunization.

L9 ANSWER 40 OF 147 MEDLINE on STN DUPLICATE 44
TI Modulation of Ia+ Langerhans cell numbers in vivo by cultured epidermis derived supernatants and by GM-CSF.

L9 ANSWER 41 OF 147 MEDLINE on STN DUPLICATE 45
TI Epidermal Langerhans cells from mice bearing a granulocyte macrophage-colony stimulating factor-producing mammary tumor display impaired accessory functions.

L9 ANSWER 42 OF 147 MEDLINE on STN DUPLICATE 46
TI Human dendritic Langerhans cells generated in vitro from CD34+ progenitors can prime naive CD4+ T cells and process soluble antigen.

L9 ANSWER 43 OF 147 MEDLINE on STN DUPLICATE 47
TI Functional studies of major histocompatibility class II-positive dendritic cells and resident tissue macrophages isolated from the rat iris.

L9 ANSWER 44 OF 147 MEDLINE on STN DUPLICATE 48
TI Relative roles of T cells and macrophages in cytokine-mediated functional transformation of cultured splenic dendritic cells.

L9 ANSWER 45 OF 147 MEDLINE on STN DUPLICATE 49
TI Expression of GM-CSF receptor by Langerhans' cell histiocytosis cells.

L9 ANSWER 46 OF 147 MEDLINE on STN DUPLICATE 50
TI In situ expression of activation markers by Langerhans' cells containing GM-CSF.

L9 ANSWER 47 OF 147 MEDLINE on STN DUPLICATE 51
TI Synergistic interaction between c-kit ligand (SCF), GM -CSF and TNF promotes optimal dendritic Langerhans cell proliferation from primitive progenitors in human bone marrow.

L9 ANSWER 48 OF 147 MEDLINE on STN DUPLICATE 52
TI Interleukin 10 inhibits T cell alloreaction induced by human dendritic cells.

L9 ANSWER 49 OF 147 MEDLINE on STN DUPLICATE 53
TI [Current data on epidermal Langerhans cells]. Donnees recentes sur la cellule de Langerhans epidermique.

L9 ANSWER 50 OF 147 MEDLINE on STN DUPLICATE 54
TI [Cutaneous immune system]. Le systeme immunitaire cutane.

L9 ANSWER 51 OF 147 MEDLINE on STN DUPLICATE 56
TI Detection of GM-CSF in the sera of children with

Langerhans' cell histiocytosis.

L9 ANSWER 52 OF 147 MEDLINE on STN DUPLICATE 57
TI Evidence that granulocyte macrophage-colony-stimulating factor regulates the distribution and differentiated state of dendritic cells/Langerhans cells in human lung and lung cancers.

L9 ANSWER 53 OF 147 MEDLINE on STN DUPLICATE 58
TI Immunohistochemical detection of granulocyte/macrophage colony-stimulating factor in Langerhans' cell histiocytosis.

L9 ANSWER 54 OF 147 MEDLINE on STN DUPLICATE 59
TI TNF and GM-CSF dependent growth of an early progenitor of dendritic Langerhans cells in human bone marrow.

L9 ANSWER 55 OF 147 MEDLINE on STN DUPLICATE 61
TI Cyclosporin increases granulocyte/macrophage colony-stimulating factor (GM-CSF) activity and gene expression in murine keratinocytes.

L9 ANSWER 56 OF 147 MEDLINE on STN DUPLICATE 62
TI GM-CSF and TNF-alpha cooperate in the generation of dendritic Langerhans cells.

L9 ANSWER 57 OF 147 MEDLINE on STN DUPLICATE 63
TI Induction of inflammatory cytokines in murine keratinocytes upon in vivo stimulation with contact sensitizers and tolerizing analogues.

L9 ANSWER 58 OF 147 MEDLINE on STN DUPLICATE 64
TI Granulocyte-macrophage colony-stimulating factor-dependent growth and erythropoietin-induced differentiation of a human cell line MB-02.

L9 ANSWER 59 OF 147 MEDLINE on STN DUPLICATE 65
TI Granulocyte macrophage--colony-stimulating factor (GM-CSF) decreases CD1a expression by human Langerhans cells and increases proliferation in the mixed epidermal cell-lymphocyte reaction (MELR).

L9 ANSWER 60 OF 147 MEDLINE on STN DUPLICATE 66
TI Properties of lymph-borne (veiled) dendritic cells in culture. I. Modulation of phenotype, survival and function: partial dependence on GM-CSF.

L9 ANSWER 61 OF 147 MEDLINE on STN DUPLICATE 67
TI Interleukin 1 binds to specific receptors on human keratinocytes and induces granulocyte macrophage colony-stimulating factor mRNA and protein. A potential autocrine role for interleukin 1 in epidermis.

L9 ANSWER 62 OF 147 MEDLINE on STN DUPLICATE 68
TI Cytokines amplify the function of accessory cells.

L9 ANSWER 63 OF 147 MEDLINE on STN DUPLICATE 69
TI The sensitization phase of T-cell-mediated immunity.

L9 ANSWER 64 OF 147 MEDLINE on STN
TI Dendritic cells and tumor specific immunity.

L9 ANSWER 65 OF 147 MEDLINE on STN
TI The 69th annual meeting symposium. II: Mechanism of necrotizing granuloma formation and its function.

L9 ANSWER 66 OF 147 MEDLINE on STN
TI Immunohistologic study of the nasal mucosa with reference to Langerhans cells.

L9 ANSWER 67 OF 147 MEDLINE on STN

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TI   Cytokine pattern of Langerhans cells isolated from murine epidermal cell
     cultures.

L9   ANSWER 68 OF 147      MEDLINE on STN
TI   Influence of microenvironmental factors on human Langerhans cell function
     in vitro.

L9   ANSWER 69 OF 147      MEDLINE on STN
TI   The binding of antigen presenting cells to T lymphocytes.


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L1          220 S (STEM(A)CELL)(S)(STIMULATOR OR DIFFERENTIATOR) AND PD<=200404
L2          103 DUP REM L1 (117 DUPLICATES REMOVED)
L3            2 S L2 AND (BETA(A)CELL OR LANGERHAN?)
L4           16 S (STEM(A)CELL)(S)(RECRUITING(3A)FACTOR) AND PD<=20040415
L5            7 DUP REM L4 (9 DUPLICATES REMOVED)
L6          844 S (BETA(A)CELL OR LANGERHAN?) (L)(EPO OR GM-CSF OR SCF OR G-CSF
L7          300 S (BETA(A)CELL OR LANGERHAN?)(S)(EPO OR GM-CSF OR SCF OR G-CSF)
L8            0 S ((BETA(A)CELL OR LANGERHAN?)(S)REGENERATION) (S)(EPO OR GM-CS
L9          147 DUP REM L7 (153 DUPLICATES REMOVED)

=> S (BETA(A)CELL (L)(EPO OR GM-CSF OR SCF OR G-CSF) AND pd<=20040415
UNMATCHED LEFT PARENTHESIS '(BETA'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> S (BETA(A)CELL)(L)(EPO OR GM-CSF OR SCF OR G-CSF) AND pd<=20040415
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   1 FILES SEARCHED...
   3 FILES SEARCHED...
L10          95 (BETA(A) CELL)(L)(EPO OR GM-CSF OR SCF OR G-CSF) AND PD<=2004041
                5

=> Dup Rem L10
PROCESSING COMPLETED FOR L10
L11          39 DUP REM L10 (56 DUPLICATES REMOVED)
                ANSWERS '1-22' FROM FILE MEDLINE
                ANSWERS '23-28' FROM FILE BIOSIS
                ANSWERS '29-37' FROM FILE CAPLUS
                ANSWERS '38-39' FROM FILE EMBASE

=> D Ti L11 1-39

L11  ANSWER 1 OF 39     MEDLINE on STN                  DUPLICATE 1
TI   Increased islet antigen presentation leads to type-1 diabetes in mice with
     autoimmune susceptibility.

L11  ANSWER 2 OF 39     MEDLINE on STN                  DUPLICATE 2
TI   BVL-1-like VL30 promoter sustains long-term expression in erythroid
     progenitor cells.

L11  ANSWER 3 OF 39     MEDLINE on STN                  DUPLICATE 4
TI   Treatment of insulin resistance in uremia.

L11  ANSWER 4 OF 39     MEDLINE on STN                  DUPLICATE 7
TI   Granulocyte macrophage-colony stimulating factor (GM-CSF) recruits immune
     cells to the pancreas and delays STZ-induced diabetes.

L11  ANSWER 5 OF 39     MEDLINE on STN                  DUPLICATE 8
TI   Altered dendritic cells (DC) might be responsible for regulatory T cell
     imbalance and autoimmunity in nonobese diabetic (NOD) mice.

L11  ANSWER 6 OF 39     MEDLINE on STN                  DUPLICATE 9
TI   A defect in bone marrow derived dendritic cell maturation in the
     nonobesediabetic mouse.

L11  ANSWER 7 OF 39     MEDLINE on STN                  DUPLICATE 10
TI   Peptide-specific cytotoxicity of T lymphocytes against glutamic acid
     decarboxylase and insulin in type 1 diabetes mellitus.

L11  ANSWER 8 OF 39     MEDLINE on STN                  DUPLICATE 11
TI   Manipulation of pancreatic stem cells for cell replacement therapy.

L11  ANSWER 9 OF 39     MEDLINE on STN                  DUPLICATE 14
TI   Effects of nerve growth factor (NGF) and other fibroblast-derived growth
     factors on immature human mast cells (HMC-1).

L11  ANSWER 10 OF 39     MEDLINE on STN                 DUPLICATE 16
TI   Alteration of the CD34+ Tf-1 beta cell line profile in response to
     long-term exposure to IL-15.

L11  ANSWER 11 OF 39     MEDLINE on STN                 DUPLICATE 17
TI   Renal abnormalities in patients with sickle cell-beta thalassemia.

L11  ANSWER 12 OF 39     MEDLINE on STN                 DUPLICATE 18
TI   Monokine-producing cells predominate in the recruitment phase of NOD
     insulitis while cells producing Th1-type cytokines characterize the
     effector phase.

L11  ANSWER 13 OF 39     MEDLINE on STN                 DUPLICATE 19
TI   Effects of certain growth factors on in vitro maturation of rat fetal
     islet-like structures.
```

L11 ANSWER 14 OF 39 MEDLINE on STN DUPLICATE 20
TI Immunologic attributes of cytokine mobilized peripheral blood stem cells and recovery following transplantation.

L11 ANSWER 15 OF 39 MEDLINE on STN DUPLICATE 21
TI Demonstration of a TH1 cytokine profile in the late phase of NOD insulitis.

L11 ANSWER 16 OF 39 MEDLINE on STN DUPLICATE 22
TI Sequential production of Th1 and Th2 cytokines in response to live bacillus Calmette-Guerin.

L11 ANSWER 17 OF 39 MEDLINE on STN DUPLICATE 23
TI Kinetics and regulation of human keratinocyte stem cell growth in short-term primary ex vivo culture. Cooperative growth factors from psoriatic lesional T lymphocytes stimulate proliferation among psoriatic uninvolved, but not normal, stem keratinocytes.

L11 ANSWER 18 OF 39 MEDLINE on STN DUPLICATE 24
TI [Contribution of cytokines to inflammatory mechanisms].
La participation des cytokines au cours des mecanismes inflammatoires.

L11 ANSWER 19 OF 39 MEDLINE on STN DUPLICATE 25
TI Targeting of "T" lymphocytes against human hepatoma cells by a bispecific monoclonal antibody: role of different lymphocyte subsets.

L11 ANSWER 20 OF 39 MEDLINE on STN DUPLICATE 26
TI Granulocyte-macrophage colony-stimulating factor-dependent growth and erythropoietin-induced differentiation of a human cell line MB-02.

L11 ANSWER 21 OF 39 MEDLINE on STN DUPLICATE 27
TI Characterization of pancreatic T lymphocytes associated with beta cell destruction in the non-obese diabetic (NOD) mouse.

L11 ANSWER 22 OF 39 MEDLINE on STN
TI [Cytokines and inflammation].
Cytokines et inflammation.

L11 ANSWER 23 OF 39 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 5
TI Mono- i disaccharides: Signaling molecules regulating genes expression in yeast, plant and animal cells.
Original Title: Mono- i disacharydy: Drozdzowymi, roslinnymi i zwierzecymi czasteczkami sygnalowymi regulujacymi ekspresje genow..

L11 ANSWER 24 OF 39 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI GLP-1 induced differentiation of pancreatic ductal precursor cell line to insulin-producing cells.

L11 ANSWER 25 OF 39 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI DC-mediated immune deviation in autoimmune diabetes.

L11 ANSWER 26 OF 39 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Inhibition of GM-CSF receptor function by a splice variant of the common beta-subunit.

L11 ANSWER 27 OF 39 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI A critical role for the cytoplasmic domain of the granulocyte-macrophage colony-stimulating factor alpha receptor in mediating cell growth.

L11 ANSWER 28 OF 39 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on

STN
TI PROPERTIES OF B CELLS AND THY-1-ANTIGEN-EXPRESSING CELLS INFILTRATING RAT RENAL ALLOGRAFTS.

L11 ANSWER 29 OF 39 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 3
TI Detection of a functional hybrid receptor γc/GM-CSFRβ in human hematopoietic CD34+ cells

L11 ANSWER 30 OF 39 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 6
TI Interleukin-7 inhibits pre-T-cell differentiation induced by the pre-T-cell receptor signal and the effect is mimicked by hGM-CSF in hGM-CSF receptor transgenic mice

L11 ANSWER 31 OF 39 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 12
TI Expansion of extrathymic T cells as well as granulocytes in the liver and other organs of granulocyte-colony stimulating factor transgenic mice: why they lost the ability of hybrid resistance

L11 ANSWER 32 OF 39 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 13
TI Tec and Jak2 kinases cooperate to mediate cytokine-driven activation of c-fos transcription

L11 ANSWER 33 OF 39 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 15
TI A truncated isoform of the human β chain common to the receptors for granulocyte-macrophage colony-stimulating factor, interleukin-3 (IL-3), and IL-5 with increased mRNA expression in some patients with acute leukemia

L11 ANSWER 34 OF 39 CAPLUS COPYRIGHT 2007 ACS on STN
TI A reduced-fat diet and aerobic exercise in Japanese Americans with impaired glucose tolerance decreases intra-abdominal fat and improves insulin sensitivity but not β-cell function

L11 ANSWER 35 OF 39 CAPLUS COPYRIGHT 2007 ACS on STN
TI Inhibition of cytokine activation processes in vitro by tenidap, a novel anti-inflammatory agent

L11 ANSWER 36 OF 39 CAPLUS COPYRIGHT 2007 ACS on STN
TI Islet expression of interferon-α proceeds diabetes in both the BB rat and streptozotocin-treated mice

L11 ANSWER 37 OF 39 CAPLUS COPYRIGHT 2007 ACS on STN
TI The regulatory effect of cytokines on IL-2 production and IL-2 receptors expression of human T lymphocyte and YT cell line

L11 ANSWER 38 OF 39 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
TI Leptin and the pituitary.

L11 ANSWER 39 OF 39 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
TI IL-3, IL-5, granulocyte-macrophage colony-stimulating factor receptor α-subunit, and common β-subunit expression by peripheral leukocytes and blood dendritic cells.

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     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 10:32:53 ON 24 SEP 2007
L1           220 S (STEM(A)CELL)(S)(STIMULATOR OR DIFFERENTIATOR) AND PD<=200404
L2           103 DUP REM L1 (117 DUPLICATES REMOVED)
L3             2 S L2 AND (BETA(A)CELL OR LANGERHAN?)
L4            16 S (STEM(A)CELL)(S)(RECRUITING(3A)FACTOR) AND PD<=20040415
L5             7 DUP REM L4 (9 DUPLICATES REMOVED)
L6           844 S (BETA(A)CELL OR LANGERHAN?) (L)(EPO OR GM-CSF OR SCF OR G-CSF
L7           300 S (BETA(A)CELL OR LANGERHAN?)(S)(EPO OR GM-CSF OR SCF OR G-CSF)
L8             0 S ((BETA(A)CELL OR LANGERHAN?)(S)REGENERATION) (S)(EPO OR GM-CS
L9           147 DUP REM L7 (153 DUPLICATES REMOVED)
L10           95 S (BETA(A)CELL)(L)(EPO OR GM-CSF OR SCF OR G-CSF) AND PD<=20040
L11           39 DUP REM L10 (56 DUPLICATES REMOVED)

=> D Ibib Abs L11 1, 3,4,5,8,9,11-14,17,20,24,34,36,38

L11  ANSWER 1 OF 39     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:   2004154338     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 15048713
TITLE:              Increased islet antigen presentation leads to type-1
                    diabetes in mice with autoimmune susceptibility.
AUTHOR:             Judkowski Valeria; Krakowski Michelle; Rodriguez Enrique;
                    Mocnick Lorraine; Santamaria Pere; Sarvetnick Nora
CORPORATE SOURCE:   Department of Immunology, The Scripps Research Institute,
                    La Jolla, CA 92037, USA.
CONTRACT NUMBER:    DK54063 (NIDDK)
SOURCE:             European journal of immunology, (2004 Apr) Vol.
                    34, No. 4, pp. 1031-40.
                    Journal code: 1273201. ISSN: 0014-2980.
PUB. COUNTRY:       Germany: Germany, Federal Republic of
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
                    (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200405
ENTRY DATE:         Entered STN: 30 Mar 2004
                    Last Updated on STN: 22 May 2004
                    Entered Medline: 21 May 2004
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AB Granulocyte-macrophage colony-stimulating factor (GM-CSF) is frequently used in preclinical and clinical protocols to modulate autoimmune responses, bone marrow transplants, and recovery from immune ablative therapies. The immunological outcome of such therapies is not fully understood. We tested the hypothesis that GM-CSF would enhance the maturation of antigen-presenting cells, facilitating presentation of beta-cell autoantigens to autoreactive T cells. We found that islet expression of GM-CSF greatly enhanced disease in male mice. Islet-derived APC but not splenic APC showed markedly enhanced capacity to stimulate in vitro proliferative responses of islet-antigen-specific autoreactive T cells. In vivo transfer of CD8(+) and CD4(+) T cells demonstrate that autoreactive T cells undergo extensive division in pancreatic lymph nodes of GM -CSF-transgenic mice compared with wild-type NOD male mice. Together, the results presented here demonstrate that expression of GM-CSF in the pancreas can enhance autoimmunity in disease-susceptible mice.

L11 ANSWER 3 OF 39 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2003138248 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12653342
TITLE: Treatment of insulin resistance in uremia.
AUTHOR: Stefanovic V; Nesic V; Stojimirovic B
CORPORATE SOURCE: Institute of Nephrology and Hemodialysis, Faculty of Medicine, Nis, Serbia.. stefan@ni.ac.yu
SOURCE: The International journal of artificial organs, (2003 Feb) Vol. 26, No. 2, pp. 100-4. Ref: 36
Journal code: 7802649. ISSN: 0391-3988.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200307
ENTRY DATE: Entered STN: 26 Mar 2003
Last Updated on STN: 18 Jul 2003
Entered Medline: 17 Jul 2003

AB Insulin resistance is a characteristic feature of uremia. As long as the hyperinsulinemia adequate to overcome the insulin resistance, glucose tolerance remains normal. In patients destined to develop type 2 diabetes, the beta cell compensatory response declines, and relative, or absolute, insulin deficiency develops. At this point glucose intolerance and eventually frank type 2 diabetes occur. Insulin resistance and concomitant hyperinsulinemia are present irrespective of the type of renal disease. Several studies have confirmed that hemodialysis (HD) treatment significantly improves insulin resistance. Both CAPD and CCPD are shown to improve insulin resistance in uremic patients. Comparing the effect of PD and HD treatment, it was found that the CCPD group has significantly higher insulin sensitivity than the HD group with the CAPD group similar to HD. Treatment of calcium and phosphate disturbances, including vitamin D therapy, significantly reduces insulin resistance in uremia. Treatment with recombinant human erythropoietin (EPO) is an efficient way to increase hematocrit, to reverse cardiovascular problems and to improve insulin sensitivity. Angiotensin-converting enzyme inhibitors have been shown to improve insulin resistance, hyperinsulinemia and glucose intolerance in uremic patients. Thiazolidinediones (TZDs), the new insulin-sensitizing drugs, provide the proof that pharmacologic treatment of insulin resistance can be of enormous clinical benefit. The great potential of insulin resistance therapy illuminated by the TZDs will continue to catalyze research in this area directed toward the discovery of new insulin-sensitizing agents that work through other mechanisms.

L11 ANSWER 4 OF 39 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 2001700162 MEDLINE

DOCUMENT NUMBER: PubMed ID: 11748649
TITLE: Granulocyte macrophage-colony stimulating factor (GM-CSF) recruits immune cells to the pancreas and delays STZ-induced diabetes.
AUTHOR: Krakowski Michelle; Abdelmalik Robin; Mocnik Lorraine; Krahl Troy; Sarvetnick Nora
CORPORATE SOURCE: Department of Immunology, The Scripps Research Institute, 10550 North Torrey Pines Road, La Jolla, CA 92037, USA.
CONTRACT NUMBER: DK54063 (NIDDK)
SOURCE: The Journal of pathology, (2002 Jan) Vol. 196, No. 1, pp. 103-12.
Journal code: 0204634. ISSN: 0022-3417.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200202
ENTRY DATE: Entered STN: 19 Dec 2001
Last Updated on STN: 5 Feb 2002
Entered Medline: 4 Feb 2002

AB Granulocyte macrophage-colony stimulating factor (GM-CSF) is one of the most widely used growth factors for enhancing immune responses and is known to recruit and activate antigen-presenting cells (APCs). This study hypothesized that overexpression of this cytokine within the pancreatic beta-cells would recruit, expand, and activate APCs. The question was whether this would lead to tolerance or autoimmunity to pancreatic antigens. This possibility was tested by preparing transgenic mice (ins-GM-CSF) whose islets expressed murine GM-CSF. By 6-8 weeks of age, these mice developed a profound mononuclear cell infiltration that often overwhelmed the exocrine pancreas, although no changes in enzyme or hormone function were apparent. The majority of the mononuclear infiltrate within the pancreas was identified as F4/80+ macrophages. Transgenic ins-GM-CSF mice had splenomegaly due to a massive increase in the macrophage population. Additionally, mononuclear cells were found within the livers of transgenic mice, with F4/80+ cells also identified within the infiltrate, indicating that GM-CSF-activated mononuclear cells circulated to organs other than the pancreas. To assess the disease potential, this study tested whether macrophage recruitment to the pancreas might accelerate or protect the islets from diabetes. It was found that the induction of diabetes by low-dose streptozotocin (STZ) was delayed and reduced within ins-GM-CSF transgenic mice, in comparison with negative littermates. Together, these data highlight the role of GM-CSF in recruiting APCs such as macrophages. Advanced cellular infiltration does not overtly harm, and may even protect, pancreatic function, as seen with the delay in chemically induced diabetes.
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L11 ANSWER 5 OF 39 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 2002219321 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11956018
TITLE: Altered dendritic cells (DC) might be responsible for regulatory T cell imbalance and autoimmunity in nonobese diabetic (NOD) mice.
AUTHOR: Boudaly Sarah; Morin Joelle; Berthier Rolande; Marche Patrice; Boitard Christian
CORPORATE SOURCE: Laboratoire de Pathologie Metabolique et Hormonale du Developpement, INSERM U. 342, Hopital Saint-Vincent-de-Paul, 82 avenue Denfert Rochereau, 75014 Paris, France.. boudaly@cochin.inserm.fr
SOURCE: European cytokine network, (2002 Jan-Mar) Vol. 13, No. 1, pp. 29-37.

Journal code: 9100879. ISSN: 1148-5493.
PUB. COUNTRY: France
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200209
ENTRY DATE: Entered STN: 17 Apr 2002
Last Updated on STN: 27 Sep 2002
Entered Medline: 26 Sep 2002

AB Nonobese diabetic (NOD) mice spontaneously develop diabetes, an auto-immune disease characterized by the destruction of insulin-secreting beta-cells by autoreactive T cells. Defects in development and/or functions of dendritic cells (DC) might be critical in eliciting the auto-immune reaction to beta cells in this model. In this paper, DC differentiation in NOD mice was investigated in vitro using bone marrow-derived progenitors (BM-DC) in the presence of GM-CSF and IL-4 or spleen-derived progenitors in the presence of GM-CSF and early acting cytokines such as Flt-3L and IL-6 (SPL-DC). In both culture systems, the absolute number of NOD DC generated was strongly reduced as compared to control strains. In addition, both BM-DC and SPL-DC from NOD mice show defective differentiation into mature DC in conventional culture conditions as indicated by low expression of MHC class II and CD80 molecules among CD11c positive cells and low capacity to stimulate allogeneic T cells. However, DC achieved full maturation when exposed to LPS, except for MHC class II expression that remained decreased. Ex vivo analysis confirmed an unusual phenotype of NOD DC. Both sets of results are thus consistent with a specific defect of DC maturation in these mice.

L11 ANSWER 8 OF 39 MEDLINE on STN DUPLICATE 11
ACCESSION NUMBER: 2001419944 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11467348
TITLE: Manipulation of pancreatic stem cells for cell replacement therapy.
AUTHOR: Peshavaria M; Pang K
CORPORATE SOURCE: Ontogeny, Inc, Cambridge, Massachusetts 02138-1118, USA.. kpang@ontogeny.com
SOURCE: Diabetes technology & therapeutics, (2000 Autumn) Vol. 2, No. 3, pp. 453-60. Ref: 70
Journal code: 100889084. ISSN: 1520-9156.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 13 Aug 2001
Last Updated on STN: 13 Aug 2001
Entered Medline: 9 Aug 2001

AB The demonstration of the existence of tissue-specific adult stem cells has had a great impact on our understanding of stem cell biology and its application in clinical medicine. Their existence has revolutionized the implications for the treatment of many degenerative diseases characterized by either the loss or malfunction of discrete cell types. However, successful exploitation of this opportunity requires that we have sufficient know-how of stem cell manipulation. Because stem cells are the founders of virtually all tissues during embryonic development, we believe that understanding the cellular and molecular mechanisms of embryogenesis and organogenesis will ultimately serve as a platform to identify factors and conditions that regulate stem cell behavior. Discovery of stem cell regulatory factors will create potential pharmaceutical opportunities for treatment of degenerative diseases, as well as providing critical knowledge of the processes by which stem cells can be expanded in vitro,

differentiated, and matured into desired functional cells for implantation into humans. A well-characterized example of this is the hematopoietic system where the discovery of erythropoietin (EPO) and granulocyte-colony stimulating factor (G-CSF), which regulate hematopoietic progenitor cell behavior, have provided significant clinical success in disease treatment as well as providing important insights into hematopoiesis. In contrast, little is known about the identity of pancreatic stem cells, the focus of this review. Recent reports of the potential existence of pancreatic stem cells and their utility in rescuing the diabetic state now raise the same possibilities of generating insulin-producing beta cells as well as other cell types of the pancreatic islet from a stem cell. In this review, we will focus on the potential of these new developments and how our understanding of pancreas development can help design strategies and approaches by which a cell replacement therapy can be implemented for the treatment of insulin-dependent diabetes which is manifested by the loss of beta cells in the pancreas.

L11 ANSWER 9 OF 39 MEDLINE on STN DUPLICATE 14
ACCESSION NUMBER: 1998444378 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9771435
TITLE: Effects of nerve growth factor (NGF) and other fibroblast-derived growth factors on immature human mast cells (HMC-1).
AUTHOR: Welker P; Grabbe J; Grutzkau A; Henz B M
CORPORATE SOURCE: Department of Dermatology, Charite-Virchow Clinic, Humboldt-University, Berlin, Germany.
SOURCE: Immunology, (1998 Jul) Vol. 94, No. 3, pp. 310-7. Journal code: 0374672. ISSN: 0019-2805.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199810
ENTRY DATE: Entered STN: 29 Oct 1998
Last Updated on STN: 3 Mar 2000
Entered Medline: 19 Oct 1998

AB We have previously shown that fibroblast and keratinocyte supernatants up-regulate expression of mast cell characteristics in the human immature mast cell line HMC-1. This effect could not be induced in HMC-1 cells by the well-known mast cell growth factor stem cell factor (SCF), probably due to mutations of the SCF receptor c-Kit in these cells. Here we report the effects of several known fibroblast- and keratinocyte-derived growth factors, namely nerve growth factor (NGF), basic fibroblast growth factor, platelet-derived growth factor and transforming growth factor-beta, on mast cell differentiation, using HMC-1 cells as a model. NGF, at 0.1-50 ng/ml concentrations, caused a marked, dose-dependent up-regulation of tryptase, Fc epsilon RI and histamine within 10 days of culture, associated with an enhanced expression of mRNA for Fc epsilon RI and mast cell tryptase. On restriction analysis, only mast cell beta-tryptase, but not alpha-tryptase, could be demonstrated. Furthermore, the high-affinity NGF receptor (TrkA) was found at both the transcriptional and protein levels, while expression of the low-affinity NGF receptor was detectable at the mRNA level only. None of the other growth factors caused a significant alteration of the mast cell markers studied when added to HMC-1 cells at concentrations known to be biologically active in other culture systems. Immature human mast cells are thus induced to assume a more mature phenotype in vitro in response to NGF, most probably via stimulation of the high-affinity NGF receptor expressed on these cells. Besides SCF, NGF should therefore be considered as an additional mast cell growth factor that contributes to human mast cell maturation at tissue sites.

L11 ANSWER 11 OF 39 MEDLINE on STN DUPLICATE 17

ACCESSION NUMBER: 97381293 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9238625
TITLE: Renal abnormalities in patients with sickle cell-beta thalassemia.
AUTHOR: Katopodis K P; Elisaf M S; Pappas H A; Theodorou J C; Milionis H J; Bourantas K L; Siamopoulos K C
CORPORATE SOURCE: Department of Internal Medicine, Medical School, University of Ioannina, Greece.
SOURCE: Journal of nephrology, (1997 May-Jun) Vol. 10, No. 3, pp. 163-7.
Journal code: 9012268. ISSN: 1121-8428.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 8 Sep 1997
Last Updated on STN: 8 Sep 1997
Entered Medline: 28 Aug 1997

AB We examined renal abnormalities in Greek patients with sickle-cell beta thalassemia (S-beta thal). A total of 17 patients aged 16-59 years suffering from S-beta thal and 17 age- and sex-matched healthy controls were studied. In all individuals we carried out a detailed study of renal function including electrolytes in serum and urine, concentrating or diluting ability, urine acidification ability, glomerular filtration rate (GFR), and hormones [such as plasma renin activity (PRA), serum aldosterone, and erythropoietin (EPO)]. Though the GFR did not differ significantly in patients and controls, half the patients had either supranormal or subnormal values. Serum potassium and uric acid were significantly higher in patients than controls. Serum phosphorus was similar in both groups, though patients with S-beta thal had significantly lower phosphate excretion indices. All patients were unable to maximally concentrate the urine, and seven also had limited ability to maximally dilute it. Five patients had incomplete distal renal tubular acidosis. Four had mild proteinuria, and six had microalbuminuria. Serum EPO and aldosterone were higher in S-beta thal patients than controls, but there was no difference in PRA between the two groups. There was a strong correlation between hemoglobin concentration and EPO levels, which was strongest in patients with GFR < 50 ml/min. We conclude that patients with S-beta thal, like sickle-cell anemia patients, present multiple abnormalities of renal function.

L11 ANSWER 12 OF 39 MEDLINE on STN DUPLICATE 18
ACCESSION NUMBER: 97329363 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9185876
TITLE: Monokine-producing cells predominate in the recruitment phase of NOD insulitis while cells producing Th1-type cytokines characterize the effector phase.
AUTHOR: Pilstrom B; Bjork L; Bohme J
CORPORATE SOURCE: Department of Immunology, The Wenner-Gren Institute, Stockholm University, Sweden.
SOURCE: Journal of autoimmunity, (1997 Apr) Vol. 10, No. 2, pp. 147-55.
Journal code: 8812164. ISSN: 0896-8411.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 5 Aug 1997
Last Updated on STN: 5 Aug 1997
Entered Medline: 22 Jul 1997

AB Cells infiltrating the Langerhans' islets of prediabetic NOD females were isolated from 6 weeks to 6 months of age. These cells were assayed at a

single-cell level for production of eight different cytokines by intracellular immunofluorescent staining. Quiescent in vivo preactivated cells were detected by in vitro stimulation with PMA and ionomycin for 4 h. The cell recruitment phase, between 6 and 12 weeks of age, is predominated by production of the monokines IL-1alpha, IL-6, and TNF After stimulation IFN-gamma and occasional IL-10 and GM-CSF producing cells could also be observed. This cytokine pattern occurs simultaneously with increasing insulitis, and we suggest that these cytokines are important in attracting inflammatory cells to the islets and maintaining the inflammatory state. A high frequency of endocrine cells producing IL-6 during this period may denote a stress response caused by initial beta-cell destruction due to cytokines released by the inflammatory cells. During the effector phase, between 4 and 6 months, there is a characteristic Th1 cytokine profile with lymphocytes producing IL-2, IFN-gamma and TNF, supposedly TNF-beta. No IL-4 production could be detected and IL-10 was very rarely found, indicating the absence of a Th2 response. Our findings show that the effector phase in NOD insulitis is a Th1 rather than a Th2-mediated event. We also demonstrate that cytokines that may cause initial tissue destruction are produced during the recruitment of inflammatory cells.

L11 ANSWER 13 OF 39 MEDLINE on STN DUPLICATE 19
ACCESSION NUMBER: 96310456 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8740398
TITLE: Effects of certain growth factors on in vitro maturation of rat fetal islet-like structures.
AUTHOR: Oberg-Welsh C; Welsh M
CORPORATE SOURCE: Department of Medical Cell Biology, Uppsala University, Sweden.
SOURCE: Pancreas, (1996 May) Vol. 12, No. 4, pp. 334-9.
Journal code: 8608542. ISSN: 0885-3177.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199610
ENTRY DATE: Entered STN: 25 Oct 1996
Last Updated on STN: 3 Mar 2000
Entered Medline: 17 Oct 1996

AB We have previously studied the expression of protein tyrosine kinases in different preparations of insulin producing cells by polymerase chain reaction (PCR). Among the tyrosine kinases thus identified were the fibroblast growth factor receptor-4 (FGFR-4), c-Kit, the insulin-like growth factor (IGF-I) receptor, and the cytoplasmic tyrosine kinase Jak2, which associates with the activated receptor for growth hormone (GH). To elucidate the putative biological effects of the receptors identified, fetal islet-like structures were cultured in the absence or presence of the ligands to the receptors identified, namely, acidic FGF (aFGF), stem-cell factor (SCF), IGF-I, and GH, whereafter insulin and DNA contents as well as insulin secretion to the culture medium were determined. Nerve growth factor (NGF), the ligand to the tyrosine kinase receptor Trk-A, was also included. aFGF and GH were found to stimulate insulin release to the culture medium, whereas SCF augmented insulin contents/DNA as well as islet DNA contents. No effects of NGF or IGF-I were detected. Immunohistochemical studies of fetal rat pancreas showed localization of the c-Kit protein to the pancreatic ducts, whereas immuno-reactivity against FGFR-4 could be detected in both endocrine and exocrine parts of the pancreas as well as in the pancreatic ducts. It is concluded that tyrosine kinase receptors may be involved in the maturation of pancreatic beta cells.

L11 ANSWER 14 OF 39 MEDLINE on STN DUPLICATE 20
ACCESSION NUMBER: 96274089 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8673041

TITLE: Immunologic attributes of cytokine mobilized peripheral blood stem cells and recovery following transplantation.
AUTHOR: Talmadge J E; Reed E C; Kessinger A; Kuszynski C A; Perry G A; Gordy C L; Mills K C; Thomas M L; Pirruccello S J; Letheby B A; Arneson M A; Jackson J D
CORPORATE SOURCE: Department of Pathology/Microbiology, University of Nebraska Medical Center, Omaha 68198-5660, USA.
CONTRACT NUMBER: R01-CA61593 (NCI)
SOURCE: Bone marrow transplantation, (1996 Jan) Vol. 17, No. 1, pp. 101-9.
Journal code: 8702459. ISSN: 0268-3369.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199608
ENTRY DATE: Entered STN: 22 Aug 1996
Last Updated on STN: 22 Aug 1996
Entered Medline: 9 Aug 1996

AB The immunologic attributes of cytokine mobilized peripheral blood stem cell (PSC) products (n = 52) and the resulting reconstitution of the hematopoietic and immunologic system following autologous transplantation were examined in a consecutive population of non-Hodgkin lymphoma (NHL), or solid tumor patients at the University of Nebraska Medical Center. Granulocyte-monocyte colony stimulating factor (GM-CSF)-mobilized PSC products had a high frequency of monocytes (31%) and bands (15%) as compared to normal peripheral blood (PB) cells. The phenotypic analysis of the mobilized PSC product revealed that they had normal levels of CD4+ cells, an increased frequency of CD8+ cells and a corresponding decrease in the CD4+:CD8+ cell ratio as compared to the peripheral blood leukocytes (PBL) of normal individuals. PSC products also had an increase in CD34+ cells as compared to PB. Natural killer (NK) and T cell activity in the PSC products were also lower than that observed in PB. Post-transplantation there was an accelerated reconstitution of NK-cell function in the PB as compared to T cell function (PHA (phytohemagglutinin) mitogenesis) which did not return to normal by day 100 post-transplantation. We also report for the first time high levels of an irradiation resistant suppressor cell activity in the PSC product and in the PB post-transplantation. There was also a concomitant increase in CD4-, CD8-, TCR alpha/beta+ cells (phenotypic homolog of 'natural suppressor' (NS) cells) in the PB post-transplantation. The number of months of prior chemotherapy correlated with PHA response but the NS activity and frequency of CD4-, CD8- and TCR alpha/beta+ cells did not. Further, cytokine mobilization and apheresis appears to contribute to the loss of PHA responsiveness and the increased levels of suppressor cell activity.

L11 ANSWER 17 OF 39 MEDLINE on STN DUPLICATE 23
ACCESSION NUMBER: 95114128 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7529261
TITLE: Kinetics and regulation of human keratinocyte stem cell growth in short-term primary ex vivo culture. Cooperative growth factors from psoriatic lesional T lymphocytes stimulate proliferation among psoriatic uninvolved, but not normal, stem keratinocytes.
AUTHOR: Bata-Csorgo Z; Hammerberg C; Voorhees J J; Cooper K D
CORPORATE SOURCE: Immunodermatology Unit, University of Michigan, Ann Arbor 48109-0530.
SOURCE: The Journal of clinical investigation, (1995 Jan) Vol. 95, No. 1, pp. 317-27.
Journal code: 7802877. ISSN: 0021-9738.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 17 Feb 1995
Last Updated on STN: 29 Jan 1996
Entered Medline: 9 Feb 1995

AB Flow cytometric analysis of primary ex vivo keratinocyte cultures demonstrated that stem cells, (beta 1 integrin+, keratin 1/keratin 10 [K1/K10-], proliferating cell nuclear antigen [PCNA-] [Bata-Csorgo, Zs., C. Hammerberg, J. J. Voorhees, and K. D. Cooper. 1993. J. Exp. Med. 178:1271-1281]) establish such cultures. This methodology also enabled the quantitation of synchronized recruitment of these cells from G0 into G1 of the cell cycle (PCNA expression), which preceded bright beta 1 integrin expression. (beta 1 integrinbright expression has been shown to be a characteristic feature of keratinocyte stem cells in culture (Jones, P. H., and F. M. Watt. 1993. Cell. 73:713-724). Using the above assay, we determined whether lesional T lymphocytes in psoriasis could be directly responsible for the induction of the stem cell hyperproliferation that is characteristic of this disease. Indeed, CD4+ T lymphocytes, cloned from lesional psoriatic skin and stimulated by immobilized anti-CD3 plus fibronectin, promoted psoriatic uninvolved keratinocyte stem cell proliferation via soluble factors. This induction appeared to be through accelerated recruitment of stem cells from their quiescent state (G0) into cell cycle. By contrast, normal keratinocyte stem cells exhibited no such growth stimulation. Supernatants exhibiting growth induction all contained high levels of GM-CSF and gamma-IFN, low IL-3 and TNF-alpha, and variable IL-4. Only anti-gamma-IFN antibody was able to neutralize growth stimulatory activity of the supernatants on psoriatic uninvolved keratinocyte stem cells. However, because recombinant gamma-IFN alone inhibited growth in this assay, these data suggest that, in psoriasis, gamma-IFN acts cooperatively with other growth factors in the immune induction of cell cycle progression by the normally quiescent stem cell keratinocytes.

L11 ANSWER 20 OF 39 MEDLINE on STN DUPLICATE 26
ACCESSION NUMBER: 92063010 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1954374
TITLE: Granulocyte-macrophage colony-stimulating factor-dependent growth and erythropoietin-induced differentiation of a human cell line MB-02.
AUTHOR: Morgan D A; Gumucio D L; Brodsky I
CORPORATE SOURCE: Department of Neoplastic Diseases, Hahnemann University, Philadelphia, PA 19102.
CONTRACT NUMBER: CA29545 (NCI)
CA44329 (NCI)
HL33940 (NHLBI)
SOURCE: Blood, (1991 Dec 1) Vol. 78, No. 11, pp. 2860-71.
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199112
ENTRY DATE: Entered STN: 24 Jan 1992
Last Updated on STN: 3 Feb 1997
Entered Medline: 31 Dec 1991

AB Peripheral blood blasts from a patient with acute megakaryoblastic leukemia were placed into liquid cultures with recombinant growth factors. Growth, but not differentiation, was supported by interleukin-3 (IL-3) or granulocyte-macrophage colony-stimulating factor (GM-CSF

) for the first 30 days of culture. Sustained growth occurred only with GM-CSF and gave rise to the cell line MB-02, which has been in continuous culture for over 1 year. The cell line retained the surface phenotype of the leukemic megakaryoblasts except for the loss of glycoproteins Ib and IIb/IIIa, which were induced after exposure to phorbol esters. The induction of erythropoiesis occurred when GM -CSF-deprived cells were cultured with erythropoietin (Epo). Well-defined morphologic stages of differentiation ranging from primitive erythroblasts to nuclei-extruding normoblasts were seen. Transforming growth factor-beta inhibited GM-CSF- and Epo-dependent growth, but not erythroid maturation. Indirect immunofluorescence using globin chain-specific monoclonal antibodies detected fetal, but not adult hemoglobin in the uninduced cells. beta-globin was induced and gamma-globin was increased after Epo exposure. Both globin species accumulated in the developing erythrocytes until terminal differentiation. Quantitative S1 analysis of beta-like globin transcripts showed very low levels of epsilon- and beta-globin expression and high levels of gamma-globin expression in cells maintained in GM-CSF. Five days after induction with Epo, epsilon message decreased to barely detectable levels while gamma and beta transcripts increased threefold and 20-fold, respectively. This novel cell line not only retains many characteristics of the leukemic megakaryoblasts from which it was derived, but also can be induced to recapitulate apparent normal erythropoiesis.

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ACCESSION NUMBER: 2006:78916 BIOSIS
DOCUMENT NUMBER: PREV200600085657
TITLE: GLP-1 induced differentiation of pancreatic ductal precursor cell line to insulin-producing cells.
AUTHOR(S): Lee, Ji Eun; Wen, Jing; Kim, Han-Soo; Park, Setting Woo; Chung, Jae-Bock; Kang, Jin-Kyung; Song, Si Young
SOURCE: Gastroenterology, (APR 2004) Vol. 126, No. 4, Suppl. 2, pp. A529.
Meeting Info.: Digestive Disease Week/105th Annual Meeting of the American-Gastroenterological-Association. New Orleans, LA, USA. May 16 -20, 2004. Amer Gastroenterol Assoc.
CODEN: GASTAB. ISSN: 0016-5085.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 25 Jan 2006
Last Updated on STN: 25 Jan 2006

AB Backgrounds. Glucagon-like peptide-1 is an intestinal incretin hormone, derived from the processing of proglucagon, that exerts insulinotropic actions on insulin-producing pancreatic beta-cells. We established multipotential progenitor cells in the pancreas, termed YGIC cell lines that are transdifferentiated from adult pancreatic acinar cell to ductal progenitor cells. Our aims are to analyze the effects of GJ_P-1 on the transdifferentiation of the established normal rat pancreatic ductal cells and to investigate the potential role of GLP-1 for the future cell therapy using normal pancreatic duct cells as a pancreatic precursor cell. Methods : Treatment with recombinant GLP-1 was carried out in the absence of serum. YGIC cells are transfected with pBX322/GLP-1 to make inducible transfectant producing GLP-1. We identified the expression of SCF, c-kit, PDX-1, Pax-4, Pax-6, Ngn-3, NeuroD, insulin, glucagon and CFTR and by western blotting, RT-PCR and ELISA methods. Results. While Hes-1, Notch-1, neuroD, Shh, Pax6 key players of embryonic pancreas development, were not modulated by the treatment of GLP-1 ngn-3 and pdx-1 was down-regulated by GLP-1 Stem cell markers, SCF and c-kit were induced by the treatment of GLP-1, whereas CFTR, ductal marker, was down-regulated. Furthermore, GLP-1 treatment induced both insulin and glucagon in YGIC cells. GLP-1, however, failed

to modulate Glut-2 and glucokinase implying that this factor affects early events of endocrine differentiation, but not terminal differentiation in these cells. Conclusions : GLP-1 stimulates the differentiation of ductal progenitor cells into insulin-producing cells. These findings suggest a model of islet development in that pancreatic progenitor cells differentiated into pancreatic endocrine cells that express GLP-1 receptors, glucagons, PDX-1 and insulin.

L11 ANSWER 34 OF 39 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:144820 CAPLUS
TITLE: A reduced-fat diet and aerobic exercise in Japanese Americans with impaired glucose tolerance decreases intra-abdominal fat and improves insulin sensitivity but not β-cell function
AUTHOR(S): Carr, Darcy B.; Utzschneider, Kristina M.; Boyko, Edward J.; Asberry, Pamela J.; Hull, Rebecca L.; Kodama, Keiichi; Callahan, Holly S.; Matthys, Colleen C.; Leonetti, Donna L.; Schwartz, Robert S.; Kahn, Steven E.; Fujimoto, Wilfred Y.
CORPORATE SOURCE: Department of Obstetrics and Gynecology, University of Washington, Seattle, WA, 98195-6460, USA
SOURCE: Diabetes (2004), Volume Date 2005, 54(2), 340-347
CODEN: DIAEAZ; ISSN: 0012-1797
PUBLISHER: American Diabetes Association
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Lifestyle modification reduces the risk of developing type 2 diabetes and may have its effect through improving insulin sensitivity, .beta .-cell function, or both. To determine whether diet and exercise improve insulin sensitivity and/or β -cell function and to evaluate these effects over time, we quantified insulin sensitivity and the acute insulin response to glucose (AIRg) in 62 Japanese Americans (age 56.5 ± 1.3 years; mean ± SE) with impaired glucose tolerance (IGT) who were randomized to the American Heart Association (AHA) Step 2 diet plus endurance exercise (n = 30) vs. the AHA Step 1 diet plus stretching (n = 32) for 24 mo. β -Cell function (disposition index [DI]) was calculated as Si + AIRg, where Si is the insulin sensitivity index. The incremental area under the curve for glucose (incAUCg) was calculated from a 75-g oral glucose tolerance test. Intra-abdominal fat (IAF) and s.c. fat (SCF) areas were measured by computed tomog. At 24 mo, the Step 2/endurance group had lower weight (63.1 ± 2.4 vs. 71.3 ± 2.9 kg; P = 0.004) and IAF (75.0 ± 7.9 vs. 112.7 ± 10.4 cm2; P = 0.03) and SCF (196.5 ± 18.0 vs. 227.7 ± 19.9 cm2; P < 0.001) areas, greater Si (4.7 ± 0.5 vs. 3.3 ± 0.3 + 10-5 min · pmol-1 · l-1; P = 0.01), and a trend toward lower AIRg (294.9 ± 50.0 vs. 305.4 ± 30.0 pmol/l; P = 0.06) and incAUCg (8,217.3 ± 350.7 vs. 8,902.0 ± 367.2 mg · dl-1 · 2 h-1; P = 0.08) compared with the Step 1/stretching group after adjusting for baseline values. There was no difference in the DI (P = 0.7) between the groups. Si was associated with changes in weight (r = -0.426, P = 0.001) and IAF (r = -0.395, P = 0.003) and SCF (r = -0.341, P = 0.01) areas. Thus, the lifestyle modifications decreased weight and central adiposity and improved insulin sensitivity in Japanese Americans with IGT. However, such changes did not improve .beta .-cell function, suggesting that this degree of lifestyle modifications may be limited in preventing type 2 diabetes over the long term.
REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 36 OF 39 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1994:628581 CAPLUS
DOCUMENT NUMBER: 121:228581
TITLE: Islet expression of interferon-α proceeds

diabetes in both the BB rat and streptozotocin-treated mice

AUTHOR(S): Huang, Xiaojian; Hultgren, Bruce; Dybdal, Noel; Stewart, Timothy A.

CORPORATE SOURCE: Dep. of Endocrine Res., Genentech, Incorporated, South San Francisco, CA, 94080, USA

SOURCE: Immunity (1994), 1(6), 469-78
CODEN: IUNIEH; ISSN: 1074-7613

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The mechanism(s) leading to β cell dysfunction in type I diabetes has not been defined. The authors investigated whether islet expression of IFNα could be a cause of the lesions that are hallmarks of type I diabetes. Streptozotocin induces the expression of interferon-α by pancreatic islets prior to the diabetes induced by streptozotocin. Increased IFNα, induced by poly I/C or expressed from a transgene will exacerbate the diabetogenic effects of streptozotocin. In another rodent model of type I diabetes (the BB rat), islet expression of IFNα precedes lymphocytic infiltration and diabetes. As in the streptozotocin model, in the BB rats poly I/C will induce islet expression of IFNα and accelerate the onset of diabetes. These results are consistent with the hypothesis that islet expression of IFNα participates in causing type I diabetes.

L11 ANSWER 38 OF 39 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2002034180 EMBASE

TITLE: Leptin and the pituitary.

AUTHOR: Sone M.; Osamura R.Y.

CORPORATE SOURCE: Dr. R.Y. Osamura, Department of Pathology, Tokai University School of Medicine, Bohseidai, Isehara, Kanagawa 259-1193, Japan. osamura@is.icc.u-tokai.ac.jp

SOURCE: Pituitary, (2001) Vol. 4, No. 1-2, pp. 15-23.
Refs: 64
ISSN: 1386-341X CODEN: PITUF9

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 022 Human Genetics
029 Clinical and Experimental Biochemistry
003 Endocrinology

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 7 Feb 2002
Last Updated on STN: 7 Feb 2002

AB In 1994, Zhang et al. of Rockefeller University in New York reported the first successful complementary DNA (cDNA) cloning of leptin by the positional cloning method. Leptin was identified as the gene of ob/ob mouse in genetic obesity syndromes. It has very strong food intake control, and body weight and energy expenditure. The name "leptin" derived from the Greek word leptos, meaning "thin." We hereby review major advances leading to our current finding of leptin, leptin receptor and its structure, the outline of homozygote, and also influence of leptin in the pituitary. (The structure of leptin) The mouse obese gene has been localized to chromosome 6. With human leptin gene on chromosome 7q31.3, its DNA has more than 15000 base pairs and consists of three exons and two introns. For bioactivation of leptin the importance of disulfide-binding site is suggested. Human leptin which replaced the 128-th arginine with glutamine has the function of an aldosteron antagonist, which is reported to have the function of athrocytosis inhibition. The resemblance of leptin precursor of human, mouse and rat is very high, i.e., mouse and rat homology is 96% and mouse and human homology is 83%. (The structure of leptin receptor) The mutant gene, which is the cause of obesity, was shown on map on diabetic mouse (db/db) chromosome 4, and it was proven to be the same as the leptin receptor gene cloned by Tartaglia et all. Further studies have found the Zucker fatty rat (fa/fa) to be incorporated into a

linkage map of rat chromosome 5, whose region of rat is the equivalent to the region of conserved synteny of the db/db mouse gene. The leptin receptor is glycoprotein consisting of a single transmembrane-spanning component. The primary structure of leptin receptor belongs to the cytokine-class1 family, the single membrane-spanning receptor, and is highly related to the gp130 signal-transducing component of the interleukin-6 (IL-6) receptor, the granulocyte colony-stimulating factor (G-CSF) receptor, and the leukemia inhibitory factor (LIF) receptor. The leptin receptor is known to have at least six existing isoforms (Ob-Ra, b, c, d, e, f) from the difference in splicing. (Homozygote Mutation of Leptin and Leptin Receptor :Hormone Secretion Disorders) The point mutation of ob/ob mouse and the splicing mutation of db/db mouse show remarkable obesity and hyperphagia. These obesity models show a reproduction disorder with both the male and the female, and they develop with homozygote. The cause is thought to be the gonadotropin secretory abnormality in pituitary. Three family lines report the cases of this deficiency, and it is considered that the secretory abnormality in pituitary develops into hypogonadotropic. These patients show low value in plasma FSHβ (follicle stimulating hormone-β and LHβ (luteinizing hormone-β which are produced from pituitary, and the plasma GnRH (gonadotropin releasing hormone) level is also low. Furthermore, the leptin receptor deficient family line was reported in 1998, in which case only the homozygote developed. The plasma leptin concentration of normal human is about 8.0 ng/ml, and this case with leptin receptor deficiency has high value of 500-700 ng/ml, which is the equivalent to the db/db mouse. (Role of Leptin in Hypothalamus-Pituitary-Periphery Function) The role of leptin which regulates pituitary hormones suggests the promotion the GHRH (growth hormone releasing hormone) secretion in hypothalamus-pituitary axis, with the possibility of the rise in secretion of GH (growth hormone) in pituitary, i.e. effects of icv (intracerebroventricular) infusion of leptin has spontaneously stimulated GHRH, which promotes GH secretion in the normal rats. On the other hand, topical treatment of GH3 (derived from a rat pituitary GH-secreting cell line) with leptin directly inhibits cell proliferation. The obesity model animals (ob/ob, db/db, fa/fa) have equally plump body compared to the normal models, which shows signs of sufficient growth. (Localization and Functional Relevance of Leptin and Leptin Receptor in Rodents Pituitary) Aside from being the food intake inhibitor and the energy control factor, leptin takes part in controlling the pituitary hormones. Promoting the secretion of GH, PRL (prolactin), TSHβ (thyroid stimulating hormone-β, FSHβ/LHβ, and inhibiting the secretion of ACTH (adrenocorticotropic hormone) are the major changes of pituitary hormones which are brought on by leptin. The expressive localization is specific, and immunohistochemistry (IHC) method recognized leptin in granular state in FSHβ, LHβ and TSHβ positive cells. In our biochemical examination, the bulk of the expression of leptin is recognized in fraction of the secretory granule. In particular, FSHβ cells had the highest percentage rate of colocalized leptin in rat pituitary. On the other hand, leptin receptor has been reported to be found only in normal rat pituitary, human pituitary adenoma, and respective cell lines in pituitaries by the RT-PCR method until now, but we disclosed for the first time the localization of leptin receptor on the plasma membrane of GH-secreting cells with the IHC method that has not been cleared so far. These findings show that leptin and leptin receptor have been expressed in different cells, and that the rat pituitary glands entertain paracrine mechanism between leptin (FSHβ/LH β cells) and leptin receptor (GH cells). The function of paracrine in this pituitary suggests a new point of view in hypothalamus-pituitary axis, and it shall be concerned with many aspects such as hormone secretions and proliferation/inhibition. (Human Pituitary Adenoma) Preliminary report of leptin and leptin-receptor relationship with pituitary adenoma that has secretion abnormality has been filed, and its manifestation is being observed by the RT-PCR. Leptin and leptin receptor are expressed in most adenoma, and it is thought to function by autocrine and paracrine pathway in the adenomas. Leptin has been located in

ACTH-secreting adenoma most frequently, especially in ACTH carcinoma. The leptin receptor is detected in all adenomas with high percentage rate, with both long and short forms, and then many cases of nonfunctioning pituitary adenomas, compared with other adenomas, have been reported to be positive with both long and short forms of leptin receptor as detected by RT-PCR. The HP75 cell line is derived from the nonfunctioning pituitary adenoma, which produces FSHβ and LHβ. The expression of leptin receptor in nonfunctioning pituitary adenoma, and the suppression of HP75 multiplication may lead to the possible hypothesis of leptin becoming one factor for the treatment of pituitary adenoma, especially in gonadotropin adenomas.

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                  patents
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               AND CURRENT DISCOVER FILE IS DATED 19 SEPTEMBER 2007.

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=> S Diabetes(S)(Stem(A)Cell) AND pd<=20040415
   2 FILES SEARCHED...
L1          449 DIABETES(S)(STEM(A) CELL) AND PD<=20040415

=> S Diabetes(S)(Stem(A)Cell)(S)G-CSF AND pd<=20040415
   2 FILES SEARCHED...
   3 FILES SEARCHED...
L2            1 DIABETES(S)(STEM(A) CELL)(S) G-CSF AND PD<=20040415

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ACCESSION NUMBER:         2004:308361  CAPLUS
DOCUMENT NUMBER:          140:297529
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TITLE: Adult bone marrow-derived stem cells for treating a diabetic condition
INVENTOR(S): Hussain, Mehboob
PATENT ASSIGNEE(S): New York University, USA
SOURCE: PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004030628	A2	20040415	WO 2003-US31116	20031002 <--
WO 2004030628	A9	20040527		
WO 2004030628	A3	20041104		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003277205	A1	20040423	AU 2003-277205	20031002
US 2004136969	A1	20040715	US 2003-676261	20031002
PRIORITY APPLN. INFO.:			US 2002-415091P P	20021002
			WO 2003-US31116 W	20031002

AB The invention provides a method for treating a diabetic condition in a mammal by administering autologous or non-autologous bone marrow, or an effective subpopulation thereof. The invention also provides a method for stimulating the mobilization and differentiation of bone marrow derived cells into pancreatic islet cells.

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